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Archives of Neurology and Psychiatry

Vol. 4 JULY, 1920 No. 1

THE NEUROSES OF PEACE*

JAMES HENDRIE LLOYD, M.D.
PHILADELPHIA

In retiring from this chair, with thanks for the honor accorded me, I have thought it worth while not so much to review the past as to forecast the future. Nevertheless, the work of the Society for the past year is worthy of mention, for it has been both active and productive, so much so that I make bold to suggest that we might with advantage add one more monthly meeting to our list, in order the better to give vent to our somewhat pent up energies. Our best enterprise was our excursion to Cape May to study the war cases of Dr. Frazier and Dr. Ingham, before which we had had a special program devoted to the military experiences of others of our members.

We have, in fact, had our minds much fixed on the neuroses of war, until, I believe, the time has come for us to turn the page and to begin to think about the neuroses of peace. It is apparent that the dangers of peace may be even greater than the dangers of war, and the panorama of the world—as spread before us today—may make the timid exclaim with King Pyrrhus, "Another such victory, and we are lost." These world phenomena appeal to us not as politicians but as physicians, not as partisans but as scientists; it is for us to discover the pathologic in them, for even in the mad whirl of pandemic hysterias in which so many others are losing their heads, the neurologists may be trusted to keep theirs on their shoulders. Let us gather the fruits of a disastrous victory, for sometimes the gleaning of Ephraim is better than the vintage of Abiezer.

The condition in which the world finds itself imposes an obligation on such a society as ours. It should aim to take a lead in sane thinking; to oppose with a persistent and trenchant criticism the vagaries of thought in high and low places: to stem the turbid tide which bears on its bosom the flotsam and jetsam of a neurasthenic age.

A recent writer in *Harper's Magazine* exults in the thought that this age and country is that of the commonplace. In his opinion nothing above a low average level is to be, or should be, allowed to survive.

* Presidential Address before the Philadelphia Neurological Society, Jan. 23, 1920.

E.N.S. 4-7-22

This writer represents the class consciousness of the mob. His millennium would be a sort of monkey paradise; but his ideas are, of course, in direct opposition to all the teachings not only of history but also of biologic science. If anything has been proved it is that progress takes place by conflict and the survival of the fittest. He and other Philistines like him are the children of the devil, not for any strength they have in them, for they have no strength; but for their very weakness, their inanity, their neurasthenic outlook and example. Many of them will procreate the specimens of dementia praecox who will form the clinical material for a future generation of Philadelphia neurologists.

REACTIONS OF WAR

For what do we see in Europe today? Old empires which for centuries had held Europe together in the bonds of some sort of civilization, are shattered into bits; and in their places is a saturnalia of riot under the aegis of certain light-hearted political philosophers who proclaim that there must be a self-determination of small peoples. These small peoples proceed to cut each others throats, determined only that nothing shall survive above the lowest level of the lowest proletariat. This new freedom has thus brought about a crisis, the ultimate effects of which no man can foresee; but for the neurologists (who are *not* politicians) it means larger hospitals, larger insane asylums, more cranks, more psychasthenics, more political assassins, more medicolegal trials in our criminal courts, more experts and more fees. I am not a pessimist, for I regard pessimism as the cheapest kind of threadbare philosophy, but it is better than the superficial optimism which is the bane of much of our public thinking in America.

Dean Inge, of St. Paul's Cathedral, London, in a recent sermon, has advanced a startling theory. It is no less than this, that western civilization has received a mortal wound in the late war. His idea seems to be that the human race can no longer bear the burden of our hard, mechanical and industrial civilization. He puts it with brutal frankness. The mass of our city population finds industry and life under twentieth century conditions repellant and unendurable, and is going to refuse to continue to produce. Men were not made for this sort of thing. It has grown to be monstrous and clean out of hand. It incarcerates men in factories and mines under abnormal conditions; it herds them in narrow streets and unhygienic dwellings; it gives them unsatisfying wages for a life of industrial slavery. Dean Inge does not say so, but it is a fact that civilized man is not far removed in point of time from the Stone Age. If you scratch him skin deep, you will find the primitive savage. If you will read Lyell's "Antiquity of Man," or

Osborne's "Men of the Old Stone Age," you will be impressed with something like a shock to find that the period of man's civilization, since the earliest records in Egypt and Babylonia, is but as yesterday compared with the long prehistoric period during which the human race existed in Europe as the contemporary of the cave-bear, the cave-hyena and the rhinoceros. Man goes back to the Pliocene period, and it is estimated that 100,000 years will not cover his career. Out of all that squalor, when he wore a piece of skin about his loins, fed on oysters and shell-fish, and fought for his cave with the hyena and the bear, he is called on in a comparatively brief period, without time for adjustment to his new environment, to endure the grievances of a highly complex and relentless civilization. Here arise some profound biologic questions. The history of evolution seems to prove, as you can read in Darwin and Haeckel, that when any species (high or low) is suddenly subjected to a new environment to which it cannot adjust itself, it invariably perishes from the earth. This is the history of the mastodon, the sabre-toothed tiger, the dodo and many other forms. The question arises, Is it to be the history of industrial man? Perhaps the only thing that will save him will be the exhaustion of the coal beds, for our gigantic modern system is founded on coal, and experts predict that the coal will be gone in a century or so. Within our own time the price of it has doubled. Perhaps when it is gone, mankind will have another chance to save itself, and to prove that the greatest advances of the human spirit, in the future as in the past, do not depend on the steam engine and the coal mine.

This thing is going to take a frightful toll of human brains and human nerves. The reaction is inevitable, and we see the signs of it not only in Europe but also in this country. Large portions of the population in America are in revolt. Large portions of the population of Europe are starving. Only this week the Austrian government — such as it is — is offering for sale or rent all the palaces, castles and imperial domains in the hope of attracting American millionaires to take them, and thus provide money to rescue little children from starvation. They are sending abroad pictures of these little ones in the last stages of inanition. A once proud empire thus pays the penalty for a false civilization.

The brutalizing effect of the late war was appalling. The atrocities perpetrated by some of the combatants were unparalleled in human history. The mask was flung off; the cave man returned to his own. But the shock, the scar remains. There is a sort of traumatic neurasthenia that has laid hold, not only of individuals, but of large masses of people.

PSYCHOLOGY OF THE STRIKE

Some one should write a paper on the psychology of the modern strike. One idea acting on large masses of men is its distinguishing feature. It is a manifestation of the instinct of the herd, when stampeded, to go in one direction, without heed to the dangers incurred or the obstacles to be overcome. There is also a dim instinct of self-preservation. It thus becomes an obsession, leading men to clamor for self-aggrandizement while ignorant of economic realities and indifferent to the claims of society at large. When it gathers sufficient momentum, as it can in a vast country like the United States, it becomes formidable by its mere bulk. It is pathologic because it partakes of the nature of a pandemic psychosis. It is a sort of folie communiquée —an example of mental contagion on a large scale. There is an underlying paranoid state, or a sense of persecution, which leads to acts of resentment and violence.

PROHIBITION A PANDEMIC HYSTERIA

But if we want to see a real pandemic hysteria, we have not far to go in this country; and it is one that did not take its origin in war, although the war helped it along wonderfully. Prohibition is the greatest pandemic hysteria since the Crusades, and it is not unlike the Crusades in some respects, for it makes its appeal to ethical and religious enthusiasm. But the Crusades lasted for nearly two centuries, so if there is any truth in the analogy, our bibulous friends need not look for relief at an early date. In fact, these moral movements have sometimes great longevity. Mahomet forbade wine in the Koran twelve hundred years ago, and that prohibition still stands in Mahometan countries, although it encourages the abuse of opium, and it did not prevent Omar Khayyam from singing the praises of the "old familiar juice." Prohibition in America took its origin largely as an industrial necessity; it is a genuine product of our industrial age. It is aimed largely against the abuse of alcohol by our industrial population. Its advocates are never weary of holding up the frightful example of the wage earner who spends his wages on drink and then goes home and beats his wife; and the employer of labor supports it because, as he will tell you, it promotes efficiency. Therefore, if it is good for the working man, it is good for all of us, for we are all brought down to a common level. Neurologists have little reason to defend alcohol, but neither can they view with complacency a popular movement which overwhelms everybody; which makes of us all a holocaust. Prohibition is a demonstration of the failure of the American people in self-government. Our fathers left us a liberal and rational constitution, but time has proved that it was too good for us. We must

need patch and disfigure it with a sumptuary law which would do credit to a village constabulary. Last week the newspapers announced that several thousand agents of the Revenue Department were let loose in this city to watch for moonshiners, boot-leggers and the manufacturers of hair tonics and ethereal highballs. These spies have the right to enter our houses, search our cellars and closets, and carry us off to jail if we don't look out. It remains to be seen what the effect of prohibition will be in reducing crime, poverty and insanity. As medical men we are bound to be impartial—but in a former paper I showed by statistics that the rôle of alcohol as a cause of insanity had been greatly exaggerated. Some of the figures were taken from the Pennsylvania Hospital for the Insane, and showed that among the female patients only about 6 or 7 per cent. had been sent there through the abuse of alcohol.

HYSTERIA OF SPIRITUALISM

But even in this world of doubt there is ever some hope. This is the time for us in Philadelphia to receive messages from the unseen world, and I have to apologize to this society for having neglected to secure the services of Sir Oliver Lodge. He and the Reverend Conwell could make us a great neurologic séance. But why are the messages which they receive from the spiritual world always about such trival matters? Does it need a voice from Heaven to tell a man where to find his gold pencil? I should like to question them about more weighty matters. Perhaps the spirits, if properly approached, could resolve some of our doubts in neuropathology. They might tell us, for instance, where the spirochete first gets in his work in locomotor ataxia, or clear up the mysteries of the circulation of the cerebrospinal fluid. Perhaps they could tell us about the functions of the red nucleus, or whether the corpus striatum is really a vestigial organ. But for a great British scientist to come all the way to America to inform our simpleminded people about a lot of old trumpery superstitions, makes me suspect that he is better fitted to be a dancing master of another St. Medard. His advent among us is a psychologic phenomenon. He is a sign of the times. We may yet have a witchcraft craze, right here in town, that will eclipse the historic one in Salem, and give us all an opportunity for first-hand study. I rather wish we would. These perversions of thought, these hysterias, these pandemics are wonderfully interesting if you only take them in the right way; and they would make a good year for this Society.

DECLINING BIRTH RATE

As we cast our eyes over a distracted world, we must not neglect to note the rather alarming decline in the birth rate in some civilized countries, especially in France. France has been losing population

from this cause at the rate of several hundred thousand a year, and is threatened, we are told, with extinction. Is the cause merely economic, or is it due to some occult decadence in civilized man? Neurasthenics are not good breeders—the prospect appalls them. Dr. Richet, before the Académie de Médecine in Paris, said that the cause was merely economic, and that France could increase the number of births per year from 700,000 to 2,000,000 by merely offering a premium on babies. The aberrations of thought on this subject are amazing, and quite in accord with the spirit of the times. Thus the French Chamber of Deputies has just rejected by the narrow margin of 219 to 200 votes a bill granting plural ballots according to the number of children, and the Senate was still seriously considering it. By this bill a man would have an additional ballot for every child: thus for three children three ballots; for five children five ballots, and so on. I do not know how this would work in France, but I think I know how it would work in Philadelphia. It would increase the population in the Fourth Ward, but not around Rittenhouse Square. In other words, it would tend to increase the submerged tenth, but not the better elements. This scheme to promote procreation by stuffing ballot boxes, reminds me of something that Carlyle tells us in his "French Revolution." When the terror was at its height, and popular frenzy triumphant, a cry went up (just as now) that France was losing population, whereupon queer fashions arose in women's dress — but you can read about this for yourselves in the third volume of his history. Moreover, this scheme is an attempt to establish a sort of child-suffrage (by proxy); and it may be compared with woman suffrage. It is a curious display of the doctrine that political perfection can be attained by counting noses. It also proves that freak legislation is not peculiar to America. And speaking of the French Revolution may remind us that during and after that cataclysm mental and nervous disorders were much increased.

PROBLEMS FOR THE NEUROLOGIST

Burke said that you cannot indict a whole people — neither, I suppose, can you indict a whole age. My object is merely to make an appraisement of some of the problems of the times, especially as they present themselves to the neurologist. They offer some tempting themes for this Society for the coming year, and I trust they will find a place on our programs. The labor unrest in this country alone is bound to show some psychiatric features. We are not concerned with its political or economic aspects except as these display a pathologic trend. We must also oppose the rampant idealism of the times; we must champion the realities. It is some of these realities, as they consist in shattered nerves and unbalanced minds, as they emerge

from the nebulous realms of a false idealism, that will demand our best thought. We are in danger of being swept into some millennium or other before we know it; and I know of no worse fate than to wake up some fine morning and find oneself in a millennium. It would be a sort of league of hallucinations, in which even the most expert neurologist might find it difficult to find his way. But if we are realists, we are not materialists. We do not sympathize with the perversion of esthetics to utilitarianism, with the onslaught on higher education, with the apotheosis of the mob, with the repressed emotions of the freudians, or with the expressed emotions of the parlor bolshevists. If we elect to turn away from the crude radicalism of the day, and to embrace a rational conservatism in medicine and in thought, it does not follow that we are pagans, suckled on a creed outworn.

TRIBUTE TO OSLER

There has just passed from the stage of this world a former illustrious member of this Society, and I would pause a moment in closing to pay a tribute to the memory of Osler. In our time no man has done more than he to promote the general culture of our profession. With a sane mind, a wide outlook, and a fluent pen, he was the natural foe of all philistinism and mediocrity, and the medical world is forever indebted to him in that in a materialistic age he always held up the true ideal of the physician, the scholar and the gentleman.

ELECTROMYOGRAPHIC STUDIES OF MUSCLES DURING HYSTERICAL CONTRACTION*

STANLEY COBB, M.D.
BOSTON

This short communication is written to put forward some laboratory evidence that the involuntary muscular contractions in hysterical conditions are physiologically similar to normal, conscious muscular contractions. From the clinical standpoint their similarity has, of course, been conceded for a long time. In any discussion of hysteria, however, it must be remembered that one is dealing with a complex disease, the pathology of which is psychologic; in other words, this pathology has its seat in the most highly integrated part of the organism. The question in such a piece of work as this is what difference, if any, one might find in the muscular contractions in states of normal psychobiologic integration as compared to the muscular contractions in states of hysterical malintegration.

Action-currents are slight changes of electrical potential concomitant with all forms of biologic activity. A record of the action-currents of a contracting muscle as recorded by the string-galvanometer is therefore termed an electromyogram. The string-galvanometer has been used in the study of the skeletal muscles by Piper,[1] who finds that when a muscle is voluntarily contracted a series of action-currents is recorded at a frequency of about 50 per second as long as the contraction lasts. This takes into consideration only the main waves (Hauptwellen) and not the wavelets which are superimposed on the larger waves. Recently Forbes[2] has criticized and amplified this work. By referring to either of these authors a detailed description of the methods may be obtained. In brief, the technic consists of placing over the muscle to be studied a nonpolarizable electrode which leads off the action-currents of the contracting muscle to the string of the galvanometer, the circuit being completed through another similar electrode on the skin near the first, but not over a contracting muscle. The movements of the galvanometer string are recorded by throwing a beam of light on to this string and photographing its shadow on a moving film. The method is the same as that used for making electro-

*This paper represents a part of the work done during 1919 as Dalton Scholar at the Massachusetts General Hospital.

1. Piper: Arch. f. Anat. u. Physiol. (Phys. Abth.), 1914, p. 345. Ibid.: Electrophysiologie Menschlischer Muskeles, Berlin, 1912.

2. Forbes and Gregg: Am. J. Physiol. **37**:118, 1915. Forbes and Rappleye: Am. J. Physiol. **42**:228, 1917.

cardiograms; in fact, with a few minor changes the same apparatus can be used. The technic used in these cases was the same as that used by Forbes except that in Cases 3 and 6 German silver electrodes were used instead of the nonpolarizable zinc sulphate electrodes. These are more easily applied to uncontrolled muscles, but do not give as sharp detail in the myogram.

The literature on electromyography is small, and there are no published records of spontaneous, hysterical muscular contractions. Gregor and Schilder [3] have shown the effect of fright on muscular contraction in normal subjects. They also have published electromyograms of katatonia. Apart from this, all the clinical work has been on phenomena with a more grossly organic pathology, such as hemiplegia, tabes,[4] ankle and patellar clonus [5] and chorea.[6] Wertheim Salomonson [7] has published two electromyograms of ankle clonus, one "organic" and the other "hysterical."

OBSERVATIONS ON HYSTERICAL MUSCULAR CONTRACTION

Electromyograms were made of six cases which showed hysterical muscular contractions. As is shown in the table, the patient in Case 1 had tonic contractions of the extensor muscle groups of both legs. The patient in Case 2 showed muscular cramps of short duration. The patients in Cases 3 and 4 had still shorter contractions occurring at more or less regular intervals and thus resembling tremors, while the patients in Cases 5 and 6 had typical convulsive tics, one of the sternomastoid and the other of the rectus abdominis. The last ten records are controls, the records being taken from various muscles on six normal persons.

A study of the table and a comparison of the myograms with one another, and with other published records of normal contractions, shows that the hysterical muscular contraction gives an electromyogram similar in all essential points to a voluntary, consciously controlled muscular contraction. In general, the hysterical contractions are less well sustained and less powerful, but in so far as their electrophysiology is concerned the two kinds of muscular contraction are the same.

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3. Gregor and Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat., Original **18**:195, 1913.

4. Gregor and Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat., Original **14**:359, 1912-1913.

5. Gregor and Schilder: München. med. Wchnschr. **59**:2851, 1912. Cobb: Johns Hopkins Hosp. Bull. **29**:247, 1918.

6. Cobb: Johns Hopkins Hosp. Bull. **30**:35, 1919.

7. Salomonson: Folia Neurobiol. **4**:1, 1910.

HYSTERICAL CONTRACTIONS IN AUTHOR'S CASES AND CONTROLS

Case Number	Diagnosis	Description of Muscular Movement	Muscle from Which Record Was Taken	Character of Electromyogram	Frequency of Main Waves per Second
1	Hysterical tonic contraction	Both legs were fully extended at knee and ankle by a steady tonic contraction of the extensor muscles that was only relaxed during sleep and that could only be overcome by application of great force. There was partial flexion of the thighs on the trunk and slight adduction of both legs; duration ten weeks. Cured by suggestion	Left tibialis anticus	Continuous tetanus of action-currents	68
			Left rectus femoris	Same	64
2	Hysterical muscular cramps	Every few minutes the patient had a series of muscular contractions beginning with grimacing and torticollis due to contraction of the platysma and sternomastoid, then the trunk stiffened out and rotated to the right and the great toe and foot extended dorsally. The spasms lasted about ten seconds, in any one muscle about four seconds; duration of the disease was four months. The patient improved by suggestion	Left tibialis anticus	Continuous tetanus of action-currents from three to five seconds	60
3	Hysterical tic of arm muscles	Short periodic contractions of the muscles of the right arm occurring about twice per second and lasting 0.3 of a second. The biceps flexed the elbow, and forearm flexors and pronators rotated the hand and flexed the fingers. Duration three months. Helped by suggestion, but relapses under emotional stress	Right biceps	Short periodic groups of action-currents	45
4	Hysterical tremor	When the right arm was extended there was a slow rhythmic rotation movement of the forearm and slight flexion and extension of the third, fourth and fifth fingers, with an increase of the extension at the elbow	Right triceps	Steady tetanus of action-currents with increase at time of movement	50
5	Hysterical tic of neck muscles	Spasmodic contractions of the right sterno-mastoid muscle and platysma occurring every five or ten seconds and pulling the chin downward and toward the left shoulder	Right sterno-mastoid	Short groups of action-currents occurring irregularly	55
6	Hysterical tic of abdominal muscles	Short sharp contractions of both recti abdominium at intervals of about three seconds and lasting about two fifths of a second. These began ten months ago with vomiting and retching. Helped somewhat by suggestion	Left rectus abdominis	Short groups of action-currents occurring irregularly	50
7	Normal control	Voluntary plantar flexion of foot....	Right gastrocnemius	Steady tetanus	50
8	Normal control	Voluntary grip with right hand.....	Flexor sublimis digitorum	Steady tetanus	58
9	Normal control	Voluntary grip with right hand.....	Flexor sublimis digitorum	Steady tetanus	47
10	Normal control	Voluntary grip with right hand.....	Flexor sublimis digitorum	Steady tetanus	48
11	Normal control	Voluntary grip on dynamometer with right hand	Flexor sublimis digitorum	Steady tetanus	52
12	Normal control	Voluntary grip on dynamometer with right hand	Flexor sublimis digitorum	Steady tetanus	63
13	Normal control	Voluntary contraction of biceps by flexing elbow against pressure	Right biceps	Regular steady tetanus	47
14	Normal control	Voluntary contraction of triceps by extending elbow against pressure	Right triceps	Steady sustained tetanus	65
15	Normal control	Voluntary contraction of sterno-mastoid muscle elicited by pressing chin toward left shoulder	Right sterno-mastoid	Steady tetanus with irregularities due to shift of leads	50
16	Normal control	Voluntary contraction of tibialis elicited by pressing upward with foot against a weight	Left tibialis anticus	Steady sustained tetanus	48

The frequency of the main waves[8] varies between 68 and 45 per second, the average being 56, for the hysterical patients. Among the controls the maximum frequency per second is 65 and the minimum 47, with an average of 53. This difference is not significant since the possible error in counting is large. These main waves are counted by taking a place in the record where the action-currents are running regularly and rhythmically, and measuring their duration, thus computing how many would occur per second if this regularity were constant. Where the contraction is well sustained and the series of action-currents clearly defined, the waves may be counted for a whole second. In such cases the averaging and measuring is unnecessary.

In former papers on this subject I have counted the small waves or wavelets as well as the main waves, but experience has shown that the number of these wavelets varies greatly with the technic of the record-making: if the film is run fast, more detail is seen and more wavelets appear; in slower records the wavelets cannot be seen and so are not counted. So too, the resistance of the patient affects the amplitude of the waves—if excessive, the wavelets are obscured.

OBSERVATIONS ON COARSE HYSTERICAL TREMORS

The muscular contractions in Cases 3 and 4 were periodic and thus had some clinical resemblance to coarse tremors; Case 3 might have been mistaken for paralysis agitans or a syphilitic tremor, and Case 4 for the intention tremor of multiple sclerosis. The electromyograms, however, show that the rate of both tremors is much slower and their periodicity is more irregular than the rates and periodicities found in the typical tremors associated with definite lesions of the central nervous system. In Case 3 (Fig. 3) the contractions come at the following periods measured in seconds: 0.55- 0.60- 0.48- 0.40- 0.40- 0.45- 0.50- 0.45- 0.60- 0.63- 0.80. In Case 4 (Fig. 4) the periods are 0.70- 0.60- 0.70- 0.55- 0.80 second. An electromyogram from a case of paralysis agitans is shown for comparison (Fig. 5). This was made from the gastrocnemius of a typical case in which there was a marked unilateral tremor of both arm and leg. The electromyogram shows a

8. The difference between the frequency of these main waves of the action-currents, and the rate of tremor must be kept clearly in mind. Action-currents accompany muscular activity; a single muscular twitch may have but one concomitant action-current; short groups of action-currents accompany each muscular contraction of a tremor or a clonus, and these groups appear periodically, and at a rate corresponding to the rate of the tremor. Making up any one of these periodic groups, however, are from two to a score of action-currents which have a wave length and frequency of their own. Thus, in Case 3 (Fig. 3) the rate of tremor is about 2 per second and the frequency of action-currents 45 per second.

remarkably regular periodicity (0.15- 0.17- 0.15- 0.15- 0.15- 0.17- 0.16- 0.17- 0.15- 0.15- 0.15- 0.17- 0.17- 0.17- 0.17- 0.17-) making a tremor rate of about 6.2 per second.

Clonus may also be regarded as a form of tremor, although it is not spontaneous and is only set up by stimulation of the proprioceptors. Electromyograms of ankle, patellar and wrist clonus show a typical rate of 6 or 7 per second. One published case [9] with an average rate of 6.6 per second showed one consecutive series of 160 contractions where the periods, from the beginning of one contraction to the beginning of the next, each measured 0.15 second. These electromyograms of clonus are almost identical in periodicity, regularity and rate [10] with those taken from the coarse tremors of paralysis agitans and neurosyphilis. In paralysis agitans, however, many cases show a slight continuous electrical activity of the muscle (Fig. 5.—V, V) even between the tremor contractions (Fig. 5.—C, C) which are accompanied by the large action-currents. This is especially marked in those cases in which the tremor is only present when the muscles are first put into a state of moderate tension either voluntarily or through emotional stimuli. This has also been noted for clonus.[5, 7] Wertheim Salomonson shows an electromyogram from a case of "real clonus" and compares it with the curve given by a case of "hysterical clonus." The former is clean cut and with only one or two action-currents per clonic contraction, and no electrical activity in the muscle between contractions; whereas the "hysterical" case shows a slight constant tetanus of action-currents at a frequency of 50 per second with superimposed on this a series of larger action-currents occurring synchronously with the clonic contractions, as is shown by a simultaneously made mechanical myogram. He concludes from these facts "that a voluntary contraction underlies the clonus that is sometimes found in normal persons or in hysteria patients" and that "this voluntary tetanus constitutes the mechanical condition necessary for the genesis of clonus. The muscular hypertony in cases of lateral sclerosis and allied diseases provides the mechanical condition which renders the occurrence of clonus possible. In cases of functional neuroses these mechanical conditions must be provided in another way, i. e., by active muscular tension."

These observations coincide with mine, but I do not agree that this "active muscular tension" indicates a neurotic or hysterical element. As mentioned above, some of the cases of paralysis agitans and neurosyphilis showed electromyograms of this type. In the case figured there was only a slight tremor when the leg was relaxed, but when

9. Cobb: Johns Hopkins Hosp. Bull. **29**:250, 1918.
10. Johns Hopkins Hosp. Bull. **29**: 1918, plate 35.

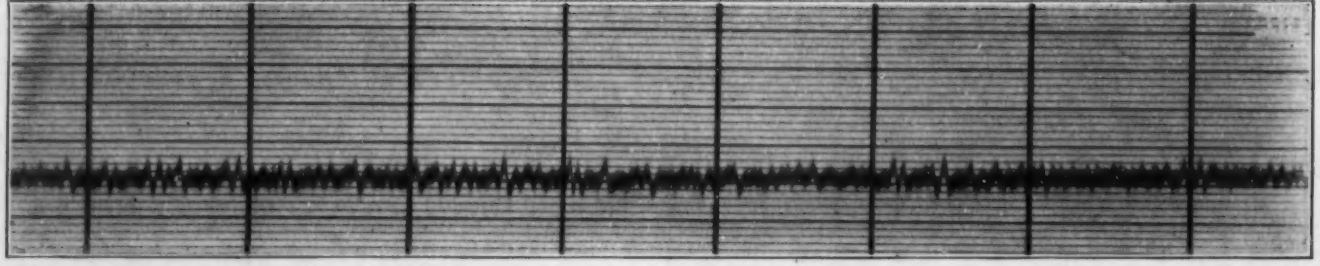

Fig. 1.—Tonic hysterical contraction: Electromyogram from left rectus femoris in Case 1, showing steady tetanus-like stream of action-currents at a frequency of 64 main waves per second. The perpendicular lines divide the illustrations into periods of ⅛ second.

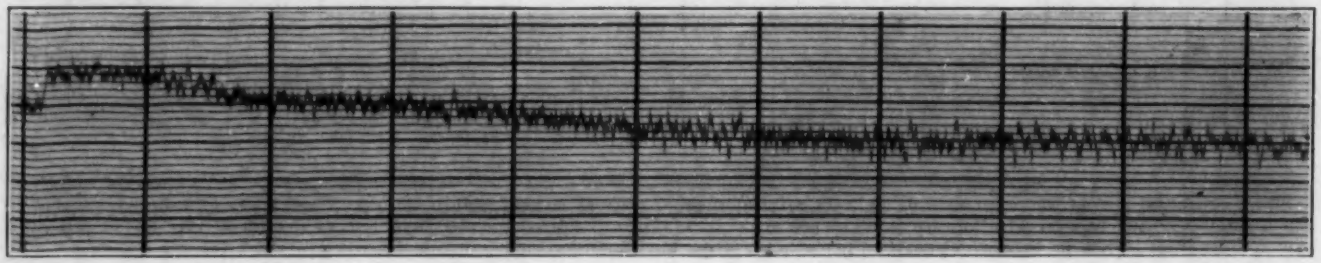

Fig. 2.—Tonic hysterical contraction: Electromyogram from left tibialis anticus in Case 2, showing a steady series of action-currents at a frequency of 60 main waves per second.

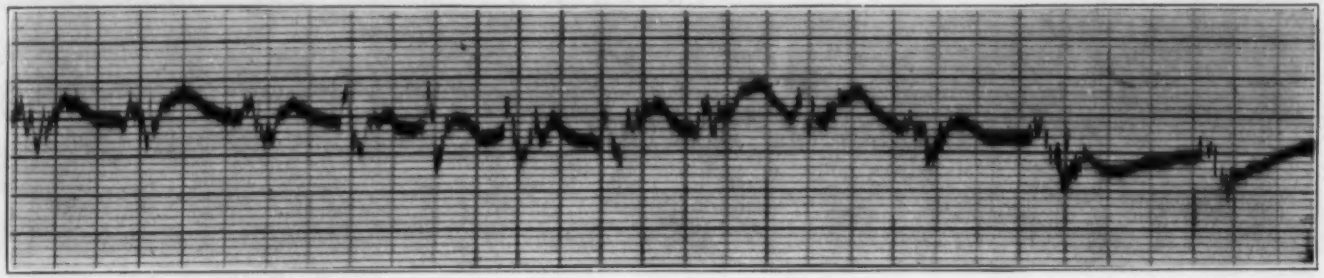

Fig. 3.—Clonic hysterical contraction: Electromyogram from Case 3. The short groups of action-currents occur periodically. The frequency of these action-currents is about 45 per second. The periods between contractions vary from 0.4 to 0.8 of a second, thus the rate averages 1.6 per second. (The plate was moving too slowly to give good detail and the focus was not sharp toward the end, necessitating retouching.)

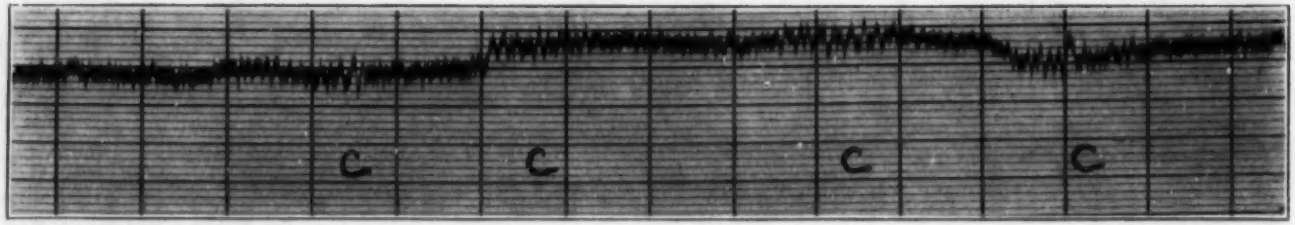

Fig. 4.—Periodic hysterical contraction: Electromyogram from Case 4. The steady stream of action-currents is due to the voluntary extension of the arm, but at points *c, c.* there is seen an increase in the size of the action-currents due to the involuntary movement, which appears at periods of from 0.58 to 0.72 of a second, i. e., at an irregular rate of 1.5 per second.

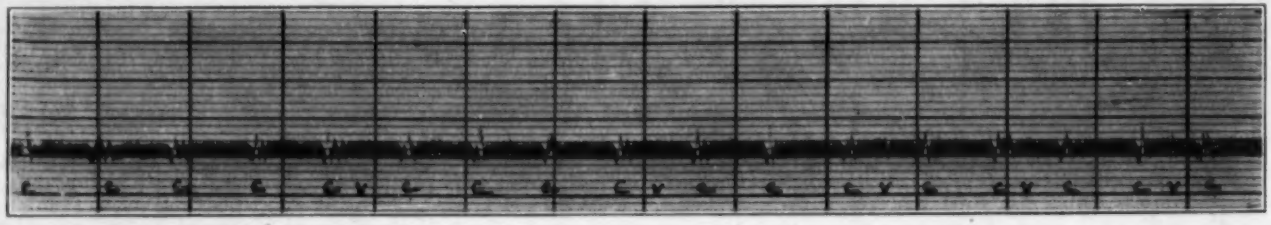

Fig. 5.—Paralysis agitans: Electromyogram from gastrocnemius. There is a regular periodicity giving a tremor rate of 6.2 per second, each tremor contraction being accompanied by a group of action-currents (*c, c, c*). Between these is a slight electrical activity (*v, v, v*) probably due to the voluntary tension put on the muscle to elicit the tremor.

the weight of the leg was balanced on the ball of the foot the tremor was brought out conspicuously (Fig. 5). In other words, a slight voluntary tension of the muscles often serves to bring out a tremor or clonus that otherwise could not be seen. Many normal people develop a clonus after taking coffee, or when fatigued,[11] by putting their muscles on just the right tension. This slight voluntary contraction underlying the grosser involuntary tremor seems, then, to depend rather on the excitability and threshold of the comparatively simple reflex arcs than on the more highly integrated psychologic levels which are concerned in the pathology of hysteria; for example, a person apparently normal and with no symptoms of hysteria may have hyperactive reflexes and clonus. Between this clonus, that needs slight voluntary muscular tension for its elicitation, and the clonus of a spastic paraplegic, there is no essential difference, for all intermediate graduations may be found. Consequently I should classify Salomonson's case not as an hysterical clonus, but as a clonus elicited in a person with hyperactive reflexes when she put her muscles on a slight voluntary tension.

In interpreting the electromyogram of a tremor, therefore, hysteria should be suspected whenever the contractions registered show conspicuous irregularity of rhythm, and when they occur with a periodicity not commonly found in the typical tremors of known clinical syndromes.

CONCLUSIONS

1. The electrophysiology of an hysterical muscular contraction is similar to that of a normal voluntary muscular contraction.

2. The short, repeated, hysterical contractions that clinically resemble tremors can be differentiated electromyographically from such a tremor as that of paralysis agitans, or from clonus, by their slower rate and greater irregularity.

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11. Levi: Arb. a. d. Neurol. Instit., Wien Univ. **2**:28, 1917.

DOUBLING OF THE SPINAL CORD*

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Variations in the gross anatomy of the spinal cord are unusual. Observation of many spinal cords, in our regular class dissections during a period of five years, tends to confirm this statement. During this period one double spinal cord was found. A careful search of the literature failed to disclose the report of a case similar to the one I describe. Although it was impossible to obtain a clinical history, the variation is so unusual as to be of more than casual interest to the clinician, the surgeon and the anatomist.

This rare variation was encountered during the regular laboratory work in the dissecting rooms at the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. Unfortunately the spinal cord was not in a state of preservation to admit microscopic study of serial sections.

The specimen was obtained from an adult white male cadaver, well nourished, about 35 years of age and of unusually good physique. A careful external examination showed no abnormalities. The vertebrae were normal and there were no abnormalities of the bony canal, either ventral or dorsal to the spinal cord. The skin and overlying tissues of the lumbar and cervical region were likewise without abnormalities.

On opening the dural sac, an unusual variation of the spinal cord and inner surface of the dura was discovered. The spinal cord was divided in its caudal portion into two distinct symmetrical halves, the two entirely enclosed in a single dural sheath as shown in the illustration. Similarly, the arachnoid stretched across as a single membrane. The bifurcation began at the caudal border of the eleventh thoracic vertebra and extended to the caudal limit of the cord, forming two independent symmetrical halves. The caudal limit of each half was at the level of the cephalic border of the first sacral vertebra. The conus medullaris of each half was independent and at the level of the third sacral vertebra, they united into a single large filum terminale, the latter coursing caudally for a distance of 5 cm. to gain attachment to the dorsal surface of the first segment of the coccyx.

The cervical and thoracic portions of the cord were normal in appearance. The peripheral nerves cephalic and caudal to the bifurcation were carefully dissected and presented no variations in the formation of the different plexuses and in their normal peripheral distribution.

The inner surface of the dura at the level of the caudal border of the twelfth thoracic vertebra likewise showed an interesting and rare anomaly. Where the division of the cord was total, a distinct band of dura was found

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*From the Daniel Baugh Institute of Anatomy of the Jefferson Medical College.

projecting between the two halves of the cord. The band of dura was directed ventrodorsal and had a first attachment of 1 cm. on both the ventral and dorsal ental surfaces of the dura. It was of an hour-glass shape and was placed in the sagittal plane. This band or fold was clearly a part of the dura, being of the same texture and consistency. In the unopened dura it seemingly caused no pressure on the halves of the cord. The division of the spinal cord began 0.5 cm. cephalic to dural band noted above.

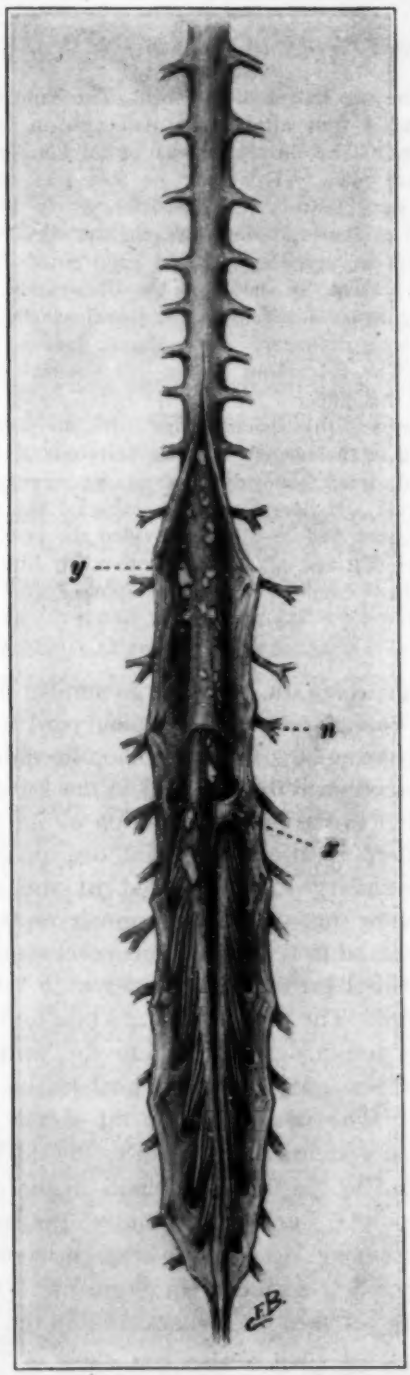
There were also present, as shown in the illustration, a number of small flattened calcareous plaques adherent to the spinal arachnoid; the largest was about 7 mm. in its long diameter. Each plaque appeared to be accompanied by a small artery. The calcareous plates will be dealt with more in detail in the discussion of this paper.

The unusual features of this specimen are: first, the presence of a bifurcated spinal cord beginning at the twelfth thoracic vertebra; second, the presence of an hour-glass-shaped dural band directed in the sagittal plane and firmly attached to the ventral and dorsal ental surfaces of the dura, which entered the dorsal median fissure and completely divided the cord; third, the absence of other abnormalities that not infrequently accompany bifurcation of the spinal cord; fourth, the extreme caudal extent of the spinal cord—first sacral vertebra.

EMBRYOLOGY

Kunitomo,[1] in a careful study of a large number of human embryos, shows that in very young specimens the spinal cord reaches the extreme tip of the tail, and throughout its length is quite uniform in structure. He further calls attention to the fact that in the formation of the filum terminale in addition to the dedifferentiation of the caudal end of the medullary tube, there is also mechanical disproportion between the growth of the medullary tube and that of the vertebral column. Streeter's studies show that the filum terminale represents that portion of the spinal cord caudal to the second coccygeal segment. This strand elongates by interstitial growth in adaptation to the ascendinng displacement of the cord. The part of the cord that undergoes dedifferention into the filum terminale is caudal to the ventriculus terminalis (the fusiform dilatation of the central canal in the lower end of the conus medullaris). The caudal tip of the dural sac maintains its initial relation to the vertebra rather than to the definitive spinal cord, and remains attached to the filum terminale in the sacral region at a more or less fixed point. Early in the embryo the spinal cord and the vertebral column lie alongside of each other in a metameric manner, corresponding in position segment for segment. Owing to the disproportion in growth between the spinal cord and the vertebral column, relative displacement of their respective levels occurs. In the first twenty-five weeks there is an ascent of the ventriculus terminalis from the level of the second coccygeal vertebra to the third lumbar, and

1. Kunitomo, K.: Contributions to Embryology, Vol. 8, Publication No. 271, Carnegie Institute of Washington **8**: 1918.



The lower part of the dural sac has been opened and turned aside. The arachnoid with the superimposed calcareous plaques is shown stretching across the spinal cord to the level of the eleventh thoracic spinal nerve indicated at N. The arachnoid below this level has been removed to show the dural plaque projecting between the halves of the spinal cord. Y indicates a calcareous plaque; N the eleventh thoracic nerve and X the dural plaque.

there remain but two segments before the adult position is reached. One may say that the principal part of the migration of the cord is accomplished during the first half of fetal life. It is thus evident that the adult position of the cord is the result of two factors — the dedifferentiation of the caudal portion of the neural tube into the filum terminale, and the disproportionate growth between the spinal cord and the vertebral column. Another factor may also enter into the formation of the filum terminale: The relation of the pia overlying the spinal cord is early established, likewise the caudal tip of the dural sac holds the same position in the embryo and the adult. It would seem plausible that as the cord ascends, due to the caudal growth of the vertebral column, the pia is gradually attenuated, interfering with the proper nutrition of the part of the spinal cord destined to become the filum terminale. The more caudal extent of the cord is, therefore, not due to a partial failure of the cord to ascend as is commonly thought, but is the result of a failure of a part of the embryonic undifferentiated cord to continue its retrogressive changes into the filum terminale.

Obviously, the length of the filum terminale depends on the caudal limit of the spinal cord. In cases in which the spinal cord extends more caudally than its usual limit, there is a corresponding decrease in the length of the filum terminale.

The neurenteric canal is a transitory communication between the neural tube and the primitive digestive tube. It is purely a temporary structure having normally completely disappeared by the end of the third week. Certain anococcygeal tumors are believed to arise from the neurenteric canal, as well as from the hind-gut. Such a canal is represented by a remnant passing from the rectum to the sacral region of the spinal canal. Keen, in 1906, reported a sacrococcygeal tumor associated with a spinal bifida and a sinus beginning at the skin, perforating the sacrum and the spinal canal, and terminating in the lumen of the gut.

The dural band present in this specimen seems to have been the factor that produced the division of the cord. I have been unable to explain the genesis of this variation on the basis of our known embryology of the cord and dura. The dural band appears to be due to a disturbance of the mesothelium resulting in an excessive mesothelial activity at this point with the projection of a fold of dura between the two halves of the cord.

LITERATURE AND DISCUSSION

Doubling of the spinal cord is a rare anomaly, and is the bifurcation of the medulla spinalis into two symmetrical or asymmetrical halves for a variable part of its course. The division usually occurs

in the lumbar or lower thoracic segments of the cord. Hanover describes a case of complete division of the spinal cord with a distinct central canal in each half extending to the fourth ventricle. Frequently this variation is only one of many anomalies in the same subject. Doubling of the spinal cord has been found associated with rachischisis, myelomeningocele, hydromeningocele and anencephalus. Frequently the anomaly is associated with cutaneous and bony malformations.

A complete doubling of the cord is one in which two well-marked symmetrical halves are present; at times one half is rudimentary and the other well developed, or the two halves are asymmetrical. The spinal cord substance collectively is probably decreased when the division of the cord is well marked. In a division that is not well-marked, or in an otherwise nondefective cord, the gray and white matter of both halves are equal to the normal amount of spinal intramedullary substance. The central canal may be absent or may be present as a distinct canal in each half, varying in outline. Thirty-five cases of double spinal cord were collected by Steiner;[2] twelve were eliminated by Bruce, McDonald and Pirie[3] as being merely split cords and thirteen as artefacts, leaving ten cases of true doubling of the cord. Of the latter, seven were associated with spina bifida. In seven of Steiner's thirty-five cases the bony vertebral canal was normal. The age of Steiner's collected cases ranges from a 3 months' old fetus to an adult of 76 years. In Steiner's cases, eight occur in adults whose ages range from 19 to 76 years. In one case the doubling was limited to the cervical and upper thoracic segments; in another (Hanover's case mentioned above) the division involved the entire cord. All other cases occurred in the lumbar region. The case described by Steiner occurred in a female infant 3½ months old. A sacral swelling was present which fluctuated and when opened revealed an oval communication with the central canal. There was likewise a medial defect of the bony vertebral column consisting of a split 2 cm. long beginning on the first sacral segment and continuing through the sacrum and coccyx. The division of the cord began at the caudal border of the fifth lumbar vertebra, the cord continuing to the level of the third sacral vertebra as two independent halves. Each half resembled a normal cord.

There are cases reported showing diffusion and spreading of the gray commissure, but without division of the white matter. These are not true and complete divisions of the cord.

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2. Steiner, F.: Ueber Verdoppelung des Ruckensmarks, Konigsburg, 1896.

3. Bruce, A.; McDonald, S., and Pirie, J. H. H.: A Case of Localized Doubling of the Spinal Cord, Rev. Neurol. & Psychiat. **111**:709, 1905. A Second Case of Partial Doubling of the Spinal Cord, ibid., 1906, IV, 6-19.

Bruce, McDonald and Pirie report a case of doubling of the cord occurring in a woman, aged 31. There were no sensory or motor symptoms. In the lumbosacral region the cord divided into two distinct trunks entirely enclosed in a single dural sheath. The division was total and a fold of dura projected for a certain distance between the two halves not reaching the ventral surface of the dural sac. The cord was completely divided for about 2 inches. There were no abnormalities of the bony vertebral canal. In the case described in this paper the fold of dura projected between the halves of the cord and had a distinct attachment to the ventral and dorsal ental surfaces of the spinal dura.

Bernstein[4] has described a case of bifurcation of the spinal cord beginning at the first lumbar segment. Examination of sections of the cord showed two seemingly complete cords united slightly along their mesial aspects. Each half of the cord presented two anterior and two posterior cornua. Caudal to the third lumbar vertebra the two halves of the cord seemed to fuse again. The filum terminale was single and large. At the level of the second lumbar segment each half exhibited a distinct central canal.

Van Gieson has called attention to the fact that artefacts are easily produced simulating a bifurcation of the spinal cord and certain cases demand the most careful study to eliminate the doubt of an artefact. This statement is confirmed by Spiller in a study of a specimen of what seemed a double spinal cord. On more careful examination and study he decided it was an artefact.

Westphal[5] has reported an unusually interesting case of a lumbar division of the spinal cord occurring in a woman, aged 29. There were no associated abnormalities. On opening the dural sac the spinal cord was found divided in its caudal portion into two distinct halves, the left half being broader and larger than the right. The dura exhibited no abnormal variations. The left half presented in its distal portion a distinct cyst which was filled with glassy mucus. The walls of the cyst were whitish in color and spotted with yellowish red areas. The latter on close examination proved to be small vessels and small hemorrhagic spots. The cervical and thoracic segments of the spinal cord were normal in appearance. Deep in the dorsal median fissure of the spinal cord numerous smooth muscle fibers were demonstrable on microscopic examination. The strands of smooth muscle increased in number caudally, blending with a distinct swelling also composed of smooth muscle. The swelling presented in its caudal portion a lumen, cylindrical in outline and forming the walls of the cyst noted above. The mass of muscle fibers and the spinal cord were covered by the overlying pia. The right side likewise presented a swelling smaller than that of the left and similarly composed of smooth muscle. Some of the nerve fibers streamed irregularly through the cyst wall. The cyst was not a dilatation of the central canal as a distinct ependymal canal was demonstrable in each half of the cord at levels with the cyst. The walls of the cyst as noted were formed of smooth muscle and microscopic examination of serial sections revealed no nervous tissue or ganglion cells.

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4. Bernstein, J. M.: Doubling of the Spinal Cord, Trans. Path. Soc. Lond. **7**:318, 1905-1906.

5. Westphal: Ueber eine bisher auscheinend nicht beschriebene. Missbildung am Ruckenmarke, Arch. f. Psychiat. **46**:712, 1906.

A number of swellings, myofibrolipoma in character, have been reported by Von Recklinghausen, Fischer, Von Ribbert and Marsh usually associated with lumbar spina bifida. In the case of Westphal the cyst was composed of smooth muscle fibers extending into the dorsal median fissure. The growth of smooth muscle fibers along the cord was first noticed by A. Pick. This interesting observation noted by Pick showed the presence of myomatous growths about blood vessels in the white and gray matter. Bonnet believes they represent a remnant of the hind-gut of the embryo occurring as an outgrowth from a rest of the hind-gut before the back is completely formed. Marchand and Von Recklinghausen show the relation between the neurenteric canal and the spina bifida. Middledorph comments on certain large intestinal tumors in the region of the coccyx as the persistence of the postanal opening. Muscatello has described a case of a fetal anomaly in which the lumen of the gut tract communicated by a winding canal directly with the central canal of the cord, confirming the observation of Marchand and Von Recklinghausen relative to the abnormal communication of the intestinal tract and the caudal portion of the ependymal canal. The latter is to be particularly remembered in spina bifida. Tillman states that growths in the coccygeal region may occur as rests of the medullary tube, the chorda, intestine, muscle and skeleton.

Mention was made in a description of this specimen of the occurrence of a number of calcareous plaques adherent to the spinal arachnoid. Calcareous deposits within the central nervous system are most common on each side of the superior longitudinal sinus as the result of calcium deposits in the arachnoidal granulations or villi (pacchionion bodies). The writer has observed the occurrence of macroscopic calcareous arachnoidal plaques in approximately 10 per cent. of all cadavers. The deposits, acording to Cushing and Weed,[6] are composed of true bone as is evidenced by the occurrence in them of the typical lacunar system, Haversian canals and the characteristic osteoblasts and osteoclasts. Another example of bony deposition is the occurrence of an osseous nodule in the falx cerebri. The observation of the dura and its reflections in our neuro-anatomy laboratory frequently show this example of bony deposit, in reality osteophytes and practically always confined to the falx cerebri. Calcareous deposits appear to be a normal physiologic process in two structures of the central nervous system, the corpus pineale and the plexus choroideus. Virchow called attention to the occurrence of lime salts in the cerebro-

6. Cushing, H., and Weed, L. H.: Studies on the Cerebrospinal Fluid and Its Pathway, No. IX. Calcareous Deposits in the Arachnoidea, Johns Hopkins Hospital Bulletin **26**:367, 1915.

spinal axis in that type of cerebral neoplasm which he designated psammome. Weed and Cushing have noted the occurrence of a thickened arachnoid covered with plaques of true bone in a patient suffering from epilepsy. He had, in early life, been a victim of severe infection.

Weed, in a study of a series of brain tumors, found a deposition of calcium salts in 40 per cent. of all cases. They have been described in all slowly-growing cerebral neoplasms. Bony growths in the form of osteophytes arising from the meninges are of frequent occurrence and have been repeatedly recorded.

In conclusion, it is interesting to comment on the possible effect of a lumbar puncture in a patient with a cord extending caudal to third or fourth lumbar vertebra. The author is not willing to venture an opinion because of the rarity of its occurrence, yet it seems a fair assumption that injury to the cord would follow with its resulting symptoms. It is not far-fetched to state that occasional untoward effects, such as paralysis following lumbar puncture, may be due to the presence of a cord extending to levels below its normal caudal limit.[7]

My thanks are due to Dr. J. Parsons Schaeffer, head of the Department of Anatomy, for permission to report this specimen and for his interest and criticism during this study.

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7. In addition to the references given, the following may be of interest: Stewart, J. P., and Bernstein, J.: A Case of Partial Doubling of the Spinal Cord, Rev. Neurol. & Psychiat. **4**:729, 1906; Streeter, Geo. L.: Factors Involved in the Formation of the Filum Terminale, Am. J. Anat. **25**: No. 1.

AN ACUTE DESCENDING RADICULAR TYPE OF EPIDEMIC ENCEPHALITIS

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INTRODUCTION

During the recurrence of epidemic influenza this winter, there have appeared a number of cases which at first seemed to be a new type of infection of the central nervous system. These have assumed a characteristic course, and should perhaps be considered a new clinical syndrome; however, from an etiologic point of view, it must of necessity be regarded as a distinct form of epidemic encephalitis. Five of these patients have come under my observation, and their recovery necessarily limits this study to the results of a clinical investigation.

Radiculitis has been described clinically, especially in conjunction with herpes zoster, syphilis, and as a secondary manifestation resulting from pressure on the dorsal roots; but as an inflammatory process progressively involving the spinal roots it is rarely encountered, and has not been described.

According to Mayer,[1] radiculitis is usually a unilateral condition, and is rarely bilateral except in the later part of its course.

Déjerine,[2] in his "Syndrome radiculaire," describes a symptomatology essentially of the nature of those noted below. He mentions the frequency of localized radiculitis in the lumbosacral and brachial segments (C 5 to C 7), and its rarity in other sections of the cord, remarking "that rarest of all it may exist in a more or less generalized form." According to Déjerine, this syndrome has for its clinical manifestations pain, hyperesthesia and paresthesia, corresponding to the dermatomes. A herpetic eruption may also be present. The deep reflexes may at first be exaggerated and later diminished, and there may be muscular atrophy and spasm of the muscles.

The clinical picture which I shall describe shows generalized distribution and presents the above "syndrome radiculaire" of Déjerine.

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1. Mayer, Edward E.: Radiculitis; Its Diagnosis and Interpretation, J. A. M. A. **71**:353 (Aug. 3) 1918.

2. Dejerine, J.: Semeiologic des affections du systeme nerveux, Paris, 1914, **2**:820 and 884.

SYMPTOMATOLOGY AND PHYSICAL SIGNS

The clinical course of this condition has been stereotyped in its symptoms, which have manifested themselves in a typical descending progression beginning in the higher cervical and terminating in the sacral region. The chief symptoms are: (1) sharp lancinating root pains, (2) paresthesia, (3) muscular spasms, (4) hyperesthesia, (5) delirium, and (6) fever.

The symptoms develop thus: The patient begins to complain of slight paresthesia in the back of the neck or shoulder; this is represented by a feeling of "threads" or a "puckered" sensation in the skin. Within twenty-four hours sharp shooting pains develop in the back of the neck or shoulders, and then progress downward into one or both arms. These pains are severe, sudden or knife-like in quality, and intermittent, suggestive of the "lightning" root pains of tabes, though of greater intensity. They prevent sleep and are relieved only by morphia.

Paresthesia of all varieties accompanies the pain, including numbness, tingling, burning, formication and "pins and needles" sensation. They are constant and distressing. An intense hyperesthesia to all sensation may develop over some one of the involved areas, perhaps in one or both arms, or, as in the case of one patient, in a small area in the suboccipital zone. This hyperesthesia usually is quite general, involving as well the anterior and posterior division of the affected zone.

Several days following the onset of pains, involuntary muscular spasms appear, causing coarse twitching movements of the head, shoulder and neck, which remind one of the spasmodic contractions occasionally occurring in spinal cord tumors. These contractions are present during sleep. One of the patients showed spasm of the diaphragm, which at first was unilateral, and later became bilateral, resulting in grunting speech. When these spasms affect the arms it is almost impossible for the patient to feed himself.

After a week the symptoms become much less severe in the arms and a mild delirium begins to develop. The mind becomes somewhat disoriented; the patient has mild hallucinosis and delusions, and shows considerable mental confusion. This acute psychotic state lasts for three or four days, followed for about two weeks by dulness and lack of interest almost to the point of apathy. No real drowsiness or lethargy was noted at any time in any of the cases.

The disease is sometimes ushered in by a chill followed by a persistent, low fever varying from 99 to 101 degrees, occasionally rising as high as 102 to 103 F. There is no acceleration of the pulse.

The clinical course of the disease is peculiar. Following symptoms in the arm and neck, it progresses downward in an orderly manner. Pain radiating in type around the upper chest develops, then girdle sensations and the tightness around waist. Pain and sensory disturbances in the arms become less severe, and spasmodic twitching movements of one or both abdominal recti or transversalis muscles appear. From the onset of symptoms until the severe arm pains cease about eight or ten days elapse. Then the symptoms confine themselves largely to the intercostal and abdominal regions for two or three days. There is generally delirium at this stage, but the pains are usually less severe. The symptoms then descend to the lower extremities; there is a tremendous recrudescence of the pain which extends down the legs with shock-like intensity, requiring an opiate for relief. Paresthesia and muscular spasms involve the legs. One patient suffered so intensely from the hyperesthesia over the sacral "saddle area" that she could hardly sit in bed. A slight increase of the fever precedes the invasion of the lumbosacral region, and after four or five days there is a return to a 99 to 100 degree temperature. The course then becomes more subacute accompanied by mild fever, a moderate number of light, fleeting root pains and some paresthesia. The patient progresses toward a slow convalescence of many weeks' duration. At this time the twitchings in the arms and legs have resolved themselves into an intention type of tremor which is coarse and oscillatory, and disappears when the patient is at rest.

At no time during the course of the disease are there any vasomotor, trophic or sensory changes other than those mentioned above. There is no anesthesia to touch, pain or temperature, no disturbance of deep sensibility, and no herpes. The reflexes are at no time profoundly altered. In the patients observed, a slight exaggeration of the deep reflexes was noted at the onset of the disease with a diminution in their activity several days after the invasion in each region. Toward the convalescent period in each case there developed some weakness of the grasp of hand and a beginning atrophy in the musculature of hands and arms, which seemed more rapid than could be accounted for by disuse. None of the patients showed alteration in pupillary reaction or any blurring of vision or oculomotor weakness. The control of the bladder and rectum was at all times retained.

One symptom of interest, noted in all patients, was an involuntary flexion of the head. The flexion was not a necessity for the patient's comfort, for motion did not increase or produce pain. This position of the head reminds one of the attitude seen in a cervical spinal cord tumor, syringomyelia, and sometimes in amyotrophic lateral sclerosis.

COURSE AND PROGNOSIS

The predominant symptoms of the syndrome justify the belief that the lesion is due to an acute descending inflammation progressively involving the dorsal roots. The onset is sudden, the pain beginning in the upper cervical region, remaining there for from nine to eleven days, then gradually descending to the chest and abdomen, remaining there for about three days. During this time the patient develops an acute psychosis. The symptoms clear above as they progress downward. The legs are next involved, and finally the sacral region. At the end of about two and a half weeks the symptoms reach a more or less stationary stage with low fever and mild, fleeting general radicular pain and paresthesia. These persist for a longer or shorter period, and the acute symptoms eventually disappear.

The convalescent stage was most characteristic in Case 4. The patients have a few pains and some paresthesia; the arms are weak, with slight muscular atrophy of the hands and arms. They are considerably fatigued and appear somewhat depressed over their long illness.

The ultimate prognosis cannot at this time be estimated, as the patients have not been observed for a sufficient period to determine the possibility of the later appearance of signs of spinal cord destruction resulting from the acute process.

LABORATORY FINDINGS

Examination of the blood revealed a moderate leukocytosis, between 12,000 and 14,000, and only a slight polynucleosis, 72 to 84 per cent. Urinary reports showed no changes, and blood cultures were negative. Spinal fluid examination in three cases revealed no increase in pressure; it was clear and colorless. The cell count was increased, 31 and 40 cells in two cases, while the third was within normal limits. All of the cells were lymphocytes, no polymorphonuclears being seen. Globulin was increased in two cases; one case showed a negative Noguchi reaction. Wassermann tests on both blood and spinal fluid were negative, and the colloidal gold curve was of the "irritation type" in two instances.

REPORT OF CASE

CASE 1.—J. McG., aged 39, elevator man, seen on Jan. 22, 1920, at St. Luke's Hospital.

Family and Past History.—This included no diseases bearing on the present condition.

Present Illness.—Following exposure ten days before (January 12) the patient first noticed intermittent sharp pain shooting down the right arm, which soon passed into the left arm. These pains, which became more frequent and intense,

were accompanied by numbness, tingling in the arms and hands, and a feeling as though the right arm were continually "asleep." Spasmodic twitching movements of the muscles of the arm and upper chest appeared soon after onset, and a spasm of the right half of the diaphragm, causing grunting respirations and a catch in the speech. The afternoon I saw him the condition was very much as described above, except that both halves of the diaphragm were twitching and the pains in the arm were beginning to abate. The temperature was 99 degrees, and an acute delirium had begun with visual hallucinations and attempts to get out of bed to go to a funeral.

Physical Examination.—The patient was a well nourished man, acutely ill, who appeared "toxic" and was delirious. The pupils reacted actively to light, were regular and equal. There was no facial asymmetry, no oculomotor palsies, and the tongue did not deviate. There were no disturbances of gait, no weakness of arms or legs, and no tenderness over nerve trunks. Reflexes: Triceps, biceps and wrist jerks were somewhat sluggish, and the abdominals were not obtained, perhaps on account of distention. Knee and ankle jerks were present, and there were no clonus or plantar extensor responses.

Sensorium: Over the posterior aspect of the neck above the seventh cervical spinous process there was a definite area of hyperesthesia extending upward to the postoccipital region; no other disturbances were noted. The temperature had varied up to this time between 99 and 100 degrees. The urine showed acetone and diacetic acid, which soon disappeared.

Spinal puncture the following day revealed 31 cells, all lymphocytes; globulin +; and the Wassermann reactions were negative here as well as in the blood.

Course of Disease.—This was most prolonged.

January 23: The patient was delirious with severe pain in the head, back of the neck and arms. He said that his pillows felt like "blocks of wood." Spasms of the abdominal muscles were first noted.

January 24: Pains were were first noticed in the legs and feet, especially in the right leg. There was mental confusion and the spasmodic twitchings were still present.

January 25 and 26: The patient complained of severe pains shooting down the legs. There was some twitching of legs and he tore the bed clothes. He obtained no relief from codein. Mentally, he appeared to be more clear. The reflexes in the arm were still sluggish. Abdominal reflexes were present. The right knee jerk was diminished; the left more active. There was no Babinski reflex or ankle clonus.

January 31: The patient was somewhat confused at times, but felt much better. No spasmodic twitchings were noted and he had infrequent mild shooting pains in the legs. No morphin has been necessary since January 29. The head was stooped. Paresthesia persisted in both arms and legs.

February 7: During the past four days he had had some slight neuralgic pains in both arms and legs, weakness of grips and slight atrophy of the forearm and hand muscles were present. Mental confusion had disappeared. Occasionally he had numbness in the extremities.

February 17: The patient sat up in a chair. He complained of pain in the right leg and back of the neck. He had profuse diaphoresis.

March 8: During the past few weeks there had been gradual progression toward recovery. He had had some pain from time to time, but it had not been distressing.

The temperature during the first week after admission was from 100 to 98 degrees; second week, from 98 to 100 degrees; third week, from 99.2 to 100.2 degrees; fourth week, 99 to 99.4 degrees; fifth week on, 98 to 99 degrees.

The urine and blood Wassermann test were negative.

The blood counts were: January 14, red blood cells, 4,400,000; hemoglobin, 93 per cent.; white blood cells, 7,200; polymorphonuclears, 70 per cent.; lymphocytes, 30 per cent. February 1: White blood cells, 14,500; polymorphonuclears, 78 per cent.; lymphocytes, 22 per cent.

The spinal fluid was examined but once, and the colloidal gold curve showed 111.51.51.510.5000.

This patient showed throughout the course of the disease more clouding of consciousness than any of the others. There was at one time almost a lethargy, but no somnolence was noted. The spasmodic twitching of his diaphragm was conspicuous; otherwise his progress was quite characteristic.

CASE 2.—F. B., aged 21, a woman, with no occupation, was seen on Jan. 29, 1920, four days after admission to St. Luke's Hospital.

Family and Past History.—This was negative, except for a chorea at the age of 6, and the presence of a hypertension which has been known for some years.

Present Illness.—The onset on January 19, was sudden, following exposure during an automobile ride the same morning. The illness began with sudden, severe, shocking and knife-like pains in the right shoulder, soon spreading down to the hands and to both shoulders. This was accompanied by paresthesia, numbness, tingling and extreme hyperesthesia to light touch. No herpes was present and the patient thought there was slight fever. Two days later spasmodic twitching movements of the muscles of the arms developed. They seemed to be initiated by the pain, and were so distressing that they prevented sleep and necessitated frequent doses of morphia. There had been an excessive amount of gas in the stomach, but no diaphragmatic spasms. In the last few days before being admitted to the hospital the hyperesthesia had left the right arm and spread upward so as to include the left posterior occipital region in which the pain was now severe, and there was a spasm of neck muscles drawing the head to the side. Paresthesia persisted in the hands and arms.

Physical Examination.—The patient was a well developed girl, apparently suffering considerable pain and complaining of lack of sleep. The head was held somewhat bowed. There was perhaps a slight lack of facial expression. There were no oculomotor or pupillary disturbances; no asymmetry of face or deviation of tongue. The lungs and heart were normal. The blood pressure was: systolic, 250; diastolic, 200. The abdomen was negative, except for slight distention. Muscular strength was good, except for some weakness of grip of both hands. Reflexes: Biceps, triceps and wrist were moderately diminished. Abdominals were present. Knee jerks and ankle jerks were exaggerated and plantar flexion was present. There were no clonus or extensor responses and no ataxia, but there was a coarse tremor of the arms and myoclonic movements of arms and of the head on the neck (trapezius and sternomastoid).

Sensorium: There was no anesthesia, and over the entire left arm extending to the midline anteriorly and posteriorly and up over occiput on left side so as to include cervical 3, 4, 5, 6, 7, 8 and T 1, 2 and 3 dermatomes, there was exquisite hyperesthesia causing the patient to wince at the touch of the finger or pin. There was no herpes.

Course of Disease.—January 27: The back of the neck, head and shoulders were painful, and paresthesia of the hands persisted.

January 30: A rise of temperature occurred from 100 to 102.3 on January 31, reaching a level of 100 degrees on February 1.

January 31: There was almost no pain in the arms, and the hyperesthesia had disappeared. She had been more comfortable for the past forty-eight hours, but there was still slight impairment of grips in the arms and some twitching. Flexion of the head was noticed. Paresthesia in the ankle, which was complained of on January 30, was increased, and there were also a few mild shooting pains down legs.

February 1: There were severe knife-like root pains in the legs with paresthesias and a hyperesthesia of the toes and balls of the feet. Twitchings of legs had also developed. She was delirious and out of bed at times. Morphin was required constantly for relief of pain.

February 2: Pain had located in the thighs and buttocks.

February 7: Great improvement had occurred during the past three days, though there were still occasional mild pains in both extremities and some in the knees.

February 10: Pains in the legs were moderate, not requiring morphia. There was intense hyperesthesia over the buttocks corresponding to sacral second, third, fourth and fifth dermatomes. The coarse tremor of the arms was still present, and strength was returning to the grip. The patient left the hospital against advice, but to date is slowly recovering.

The temperature from the first week after admission was between 100 and 102.3 degrees, the latter figure representing the apex of the rise ushering in the involvement of the legs. The temperature during the second week was between 99.2 and 101 degrees; third week, from 99.1 to 100.3.

Laboratory Findings.—The blood pressure showed the following readings: systolic, 250; diastolic, 200; systolic, 200; diastolic, 150; systolic, 172; diastolic, 130.

January 25 the urine showed a specific gravity of 1.020; acid reaction; marked trace of albumin; sugar negative; few hyaline casts and red and white blood corpuscles in moderate numbers. These findings connote a nephritis of some severity.

Blood culture was negative.

Blood Wassermann test was negative.

The blood counts were, January 25: red blood cells, 5,000,000; hemoglobin, 103 per cent.; January 28: white blood cells, 12,300; polymorphonuclears, 78 per cent.; lymphocytes, 22 per cent. White blood cells, 11,200; polymorphonuclears, 80 per cent.; lymphocytes, 20 per cent. January 30: White blood cells, 6,500; polymorphonuclears, 82 per cent.; lymphocytes, 18 per cent. February 5: White blood cells, 16,500; polymorphonuclears, 84 per cent.; lymphocytes, 16 per cent.

The spinal fluid, January 25, showed 4 cells, all lymphocytes; the globulin was negative; the Wassermann reaction was negative. Colloidal gold curve: 1110.5000000.

The spinal fluid on February 4 showed 6 cells, 100 per cent. lymphocytes. Globulin and Wassermann tests were negative. Colloidal gold curve: 1111.5 10.50000.

This patient presented the characteristic picture of this syndrome. The few signs of thoracic involvement (chest and abdomen), and a

delirium of short duration were noteworthy. Neither examination of the spinal fluid showed pleocytosis, although the colloidal gold test showed a definite irritation curve. She showed no cerebral signs, except the delirium and a certain lack of facial expression.

CASE 3.—J. B., a colored woman, aged 32, married, was admitted to the City Hospital, Feb. 2, 1920, on the medical service. She was transferred to the neurological service Feb. 3, 1920.

Chief Symptoms.—These were twitchings of the body and pain in the shoulders, chest and abdomen.

Present Illness.—This began two weeks before the patient was admitted to the hospital after she had caught cold while working in a laundry. Pain began in the back of head and neck and three days later involved the arms with severe shooting pains, requiring an opiate for relief. Accompanying the pains were definite paresthesias, such as numbness and "pins and needles" over the shoulders, arms and hands, and hyperesthesia so intense that she could not stand the touch of her clothes against her. Her arms began to twitch at this time. She was taken to a hospital and in four days improved so much that she was sent home. The pain gradually left the arm and shifted down to the chest, and two days before admission to the City Hospital involved the abdomen with radiating pain to the front and with spasms of the abdominal muscles. The pain in the legs became so severe that she again went to the hospital.

On admission she was screaming with severe intermittent lancinating pain in the legs and complaining that her legs were numb and asleep. She was delirious, crying for relief, trying to get out of bed and the window, cursing and complaining that people were hanging over the bed when no one was near.

Physical Examination.—The patient was a well nourished colored woman, apparently delirious, and suffering from intermittent pain. There was no ptosis or diplopia; the pupils reacted actively to light and were regular and equal. There was no facial asymmetry or deviation of tongue. Heart and lungs were negative. There were spasmodic twitching movements of both recti muscles occurring at intervals and a coarse almost spasmodic tremor of the hands. There were weakness of muscles and no tenderness over the nerve trunks. Reflexes were present in the arms and legs and not altered. There was no Babinski reflex, no clonus or ataxia of arms and legs.

Sensorium: Over the back from the spine of the scapulae down to the sacrum there was hyperesthesia to light, touch, and pin prick that came well forward to the abdominal walls. The temperature varied between 99 and 100 degrees, and the pulse coincided.

Owing to the noisy delirium the patient was transferred to the psychopathic ward and so passed from detailed observation, also preventing any laboratory examinations from being made. Three days later I saw her again, and at that time she had come out of the delirium, was quite oriented, but was still suffering with pain in the legs. The spasms had disappeared, and she complained that the pressure of the bed clothes on her legs and feet was painful, and of a burning feeling in the legs.

Examination at that time showed a disappearance of the hyperesthesia over the back, no twitchings in the recti muscles, a coarse tremor of the arms and slight spasmodic twitchings in the legs at times. Over both legs from the knees

to the toes on the outer aspect corresponding to dermatomes L 4 and L 5 there was intense hyperesthesia to all sensation. The temperature was 100 degrees.

No spinal puncture had been made and she felt distinctly improved. She left the hospital two days later against advice and passed from further observation.

This patient presented the syndrome of a typical descending radiculitis accompanied by mild fever and a delirium. Although her further progress was not known and no laboratory data were available, the course of the disease while she remained in the hospital was characteristic in all respects.

CASE 4.—L. B., aged 56, an Italian baker, was admitted to the Third Division of the Neurological Institute Feb. 13, 1920.

History.—The patient had always been well until four weeks before. On January 19, he suddenly felt a shock-like pain in the little finger of the left hand which traveled up the arm and then included the right arm, easing a little in the left. Arms and hands felt numb, heavy and "swollen," and there was an intense hyperesthesia of the arms, becoming so severe at the back of the neck that he could not endure the touch of the pillow against the skin. Spasmodic twitching developed in the arms, and accompanied the shooting pains. The patient suffered so much that morphin had to be administered continually. He was restless and could not sleep. After about ten days to two weeks the symptoms progressed to the chest and then a constriction about the waist was complained of with radiating pains and spasm of the recti muscles. At no time was fever noted by the family, and there was no delirium, but restlessness was a prominent feature during the first ten days. After the pain left the arms, he did not suffer; even when the paresthesia and pain progressed to the legs, he did not complain a great deal. For ten days before entering the hospital he had been sleepy and drowsy, but at no time were blurred vision, diplopia, ptosis or herpes present.

On admission he complained of pain and stiffness in the back of the neck, and his head felt heavy. There was a tremor of the hands, some weakness of both hands and arms, and scattered fleeting pain throughout legs and body.

Physical Examination.—The patient lay quietly in bed and did not look ill. There was a perceptible lack of expression about his face when in repose. External ocular movements were normal; the pupils were equal, regular and reacted to light. There was no tremor of the face. Heart, lungs and abdomen were negative. The teeth show marked pyorrhea. There was no speech disturbance. The strength of the arms was slightly impaired and the grip was a little weak. There was a coarse oscillatory tremor of the hands and arms which at times seemed to involve the legs as well. This was somewhat of an "intention" type. There appeared to be slight atrophy of the hands and forearm muscles. The reflexes in the arms were somewhat sluggish. Knee jerks and ankle jerks were exaggerated. Plantar flexion was present. No sensory disturbances were present.

The patient did not have fever.

Laboratory Examinations.—Blood and spinal fluid Wassermann tests were negative. The blood examination revealed: red blood cells, 4,600,000; hemoglobin, 83 per cent.; white blood cells, 13,000; polymorphonuclears, 73 per cent.; lymphocytes, 27 per cent.

March 9: White blood cells, 12,000; polymorphonuclears, 56 per cent.; lymphocytes, 44 per cent.

The spinal fluid showed 40 cells, all of which were lymphocytes. The globulin was positive; colloidal gold curve: 1122200000. The withdrawal of fluid caused an improvement in the patient's condition. The second examination of the sppinal fluid on March 6 showed 37 cells, 100 per cent. lymphocytes; globulin +.

Althought this patient was seen after all the acute symptoms had subsided and with no facts except those which the wife was able to give, we have, nevertheless, a typical picture of descending radiculitis, the characteristic tremor remaining and a pleocytosis in the spinal fluid. The patient was making slow progress toward a complete recovery.

CASE 5.—E., aged 15, was a schoolboy. For the privilege of reporting this case I am indebted to Dr. W. H. Glafke, who saw it in consultation, and kindly furnished me with the following data:

History.—On January 9, without any previous disease, such as influenza, he came home from work complaining of a little headache. On January 10, while in New York with his mother, he complained of slight pain in his shoulder, which he thought was worse when he rode on the subway. When he arrived home he could not take his coat off easily, and complained that he could not use his right arm. It was not really paralyzed, however, as he swung the arm back and forth "in order to stir up the blood." The next day he had severe pain through the right side of the head, back of the ear, and on the next day his right upper jaw ached and the teeth felt cold. During the following day or two the pains traveled downward into the neck, shoulder and trunk, with extreme pain running around both sides of the body. The pain was spasmodic in character and was associated with visible jerking and contraction in the trunk muscles. He was extremely restless. After four days of this the leg muscles became involved and he was constantly stretching his legs, complaining of drawing sensations and pain in them. He had had no temperature up to this time, but now he developed slight irregular fever up to 100 degrees. He became restless and noisy after being removed to the hospital, begging to be taken home. Blood tests taken then were reported by the family to be negative. After four or five days in the hospital he was removed to his home and became delirious about the twenty-first. He would speak and answer questions correctly, but then would lapse into a state of muttering delirium, complaining that the Bolsheviki were after him, and his eyes would rove about in a frightened way, apparently as the result of visual hallucinations. He could not sleep at all in spite of chloral and bromids. I (W. H. G.) saw him on the twenty-third. His temperature was running up and down from 102 degrees. He was still delirious. His pupils reacted to light and were equal. His neck was not stiff; in fact, the neck muscles were inclined to be paretic and his head dropped forward most of the time. He was not complaining of pain. The lungs, heart and abdomen were negative. He was generally very restless, and there was considerable gross tremor and twitching of all the muscles of the arm, trunk and legs. He had a generalized rash over the body (probably from bromids, and not of herpetic type). A spinal puncture was not done at any time. On January 29 his mother wrote that he was somewhat better, and was less delirious. The jerking of the

muscles had ceased, and he was complaining of pins and needles all through his body. The temperature was 101 and the pulse 120. He was beginning to get a few hours' natural sleep at a time. The attending physician felt that he was slowly recovering.

This case presents the typical syndrome of the disease with descending progression of the symptoms from above downward with accompanying delirium. An exceptional feature in this patient was the pain and paresthesia in the upper jaw and teeth, evincing an involvement of the second branch of the gasserian ganglion.

DISCUSSION

The syndrome described accompanies an acute infectious disease producing symptoms largely spinal in localization, except for the delirium. We are dealing, therefore, with a lesion chiefly affecting the meninges of the cord and especially involving the posterior roots in a progressive descending inflammation.

The appearance of these unusual cases was almost simultaneous, and their etiology was at that time puzzling and a subject for the most thoughtful speculation. It was an acute infection of the central nervous system coincident with the epidemic of influenza, but was it a new disease entity or the specific selective action of the influenza toxin on the posterior roots? A noticeable feature of the present recurrence of influenza has been the frequency of fleeting and often sharp, severe, localized types of neuralgic and radicular pains. It seemed, therefore, that this might be considered an influenza complication in spite of a negative history in each case. However, as deviations from this type appeared, the condition gradually simplified itself, and it is now quite certain that this represents only another of the many forms of epidemic encephalitis.

TYPES OF EPIDEMIC ENCEPHALITIS THAT HAVE INCLUDED A RADICULAR SYNDROME

In the preceding part of this paper I have assumed that certain cases of descending radiculitis were closely allied to, and were probably a posterior poliomyelitic form of, epidemic encephalitis. There are certain features about these cases that support this hypothesis. All the patients at one time showed, in the course of a mild febrile disease, a rather definite change in the psyche. This first manifested itself by an acute hallucinatory delirium with confusion and some disorientation, and when this condition gradually cleared up a subdued, apathetic, but not at any time a true lethargic, state remained. Three of the patients showed a decided lack of facial expression, and the tremor, though not of the "pill rolling type," nevertheless was of

a coarse, oscillating character, the whole suggesting the "parkinsonian" picture. These symptoms being characteristic of certain cases of lethargic encephalitis, we may venture to suspect a close relationship on the basis of these cases alone.

Soon after observing these cases of radiculitis with delirium, another case was seen which presented the same clinical syndrome.

The symptoms began ten days after defervescence from an influenzal pneumonia with lancinating pains in the arms, paresthesia and hyperesthesia with spasmodic twitchings and delirium. The symptoms were in every particular the same as observed in the other cases, and they progressed downward after one week in the arms, gradually giving girdle sensations about the abdomen with spasms of the recti muscles. On the ninth day, the patient complained of diplopia and somnolence, and developed sluggish pupils, bilateral ptosis, weakness of the left sixth nerve; in fact, all the classic features of an epidemic encephalitis. These symptoms progressed, and four days later death resulted (see Case 6). This case was of unusual interest, as the patient presented a definite history of an influenza with a pneumococcus group 1 pneumonia progressing to cure with development of radicular cord symptoms, and then succumbed to a typical acute epidemic encephalitis, the whole forming an apparently unmistakable link between the two conditions.

Another case that also showed this relationship ran a course of the typical radicular syndrome with delirium during the first week. The disease started in the arms, progressed in a descending course, and at the same time the patient complained of diplopia and blurring vision. There was some tendency to lethargy, while a lack of facial expression was noted by her physician. The radiculitis progressed as far downward as the inguinal region and then stopped. The diplopia remained until four weeks after, when I saw her with a left sixth palsy, slight bilateral ptosis, mask-like face, and coarse tremor of hands and feet. We have here again a case with a descending radiculitis which presents evidence of involvement of the midbrain and the nucleus lentiformis (globus pallidus), as well as the posterior spinal ganglions (see Case 8).

One other case (7) which I report in detail shows a similar antecedent descending radiculitis followed by a typical encephalitis.

CASE 6.—D. C. D., housewife, formerly a nurse, was seen on Feb. 6, 1920.

Family History.—This was negative.

Past History.—She had one boy, 2 years old. The patient had been well, was six and a half months' pregnant, and had just finished nursing her husband through a mild attack of influenza when she developed it.

Present Illness.—Her illness started with influenza on January 19. Three days later she developed signs of bronchopneumonia, which turned out to be caused by type 1 pneumococcus. Serum was given with a rapid defervescence and she was afebrile for about ten days, when on February 1 she complained of a "thread-like" feeling in the left side of the neck. This developed in twenty-four hours into a severe knife-like, shooting pain down the arms and back of the neck with such intense hyperesthesia that she could not rest on the left shoulder, nor could she bear the touch of her negligeé. Paresthesia of the arms and hands was present and spasmodic twitching of the arms, beginning in the left, and progressing to the right. The pain was so severe that no relief or rest was obtained without morphin. Forty-eight hours before I saw her she became slightly delirious; the delirium became more and more pronounced and she became somewhat excited, had hallucinations and tried to get out of bed, but she could talk quite rationally when aroused and was oriented. The following day when I saw her the patient was clear as to all the details of her long illness, but when left alone she mumbled and talked to herself, and attempted to get up. She was comfortable, only complaining of an occasional twinge of pain down her arms and a feeling as though "she had had her fingers in lime and soda," with some numbness.

Physical Examination.—This revealed that heart and lungs were normal; the face was bright; the pupils were normal; there were no cranial nerve signs and no tremor except a rare twitching of the arm. Triceps, biceps and wrist reflexes were exaggerated. Leg reflexes were normal. Sensorium: Moderate hyperesthesia over right arm; none on left; no other disturbances. Uterus was enlarged above the umbilicus; the bladder was distended. Temperature for the past week varied between 99 and 100.3 degrees. The pulse rate was 80.

Course of Disease.—February 7: She had complained of some radiating pain around the chest and on the next day about the upper abdomen, asking her husband to unfasten the night sac in the back, as it was too tight. Diplopia was noticed on that day for the first time, and objects seemed blurred. The temperature had been between 101 and 103.8 degrees. February 9: She was very stuporous and apathetic, and slept a great deal. She was definitely lethargic, but could with ease be aroused to an intelligent conversation. Twitchings of the arm and abdomen were present, and some subsultus. Catheterization had been necessary.

Physical Examination.—The patient was muttering to herself; the mental condition has been described above. There were no stiffness of neck or Kernig's sign. There was slight weakness of left sixth nerve, with lack of coordination of eye movements. The pupils were small, equal, irregular and reacted sluggishly to light. Fundi: Veins engorged, outline of nasal borders of both disks was obscured. All deep reflexes were exaggerated; there were no pathologic signs. The pulse rate was from 120 to 130; respiration, 38. Spinal puncture: pressure slightly diminished; cells, 16—all lymphocytes; globulin +. A diagnosis of epidemic encephalitis was unhesitatingly made.

February 10: The condition was more serious. The temperature was between 103.4 and 104.2 degrees; pulse, 150. The mental state was more profound, but she could be aroused. There was tremor of the arms coarse in character and there was an unquestionable lack of facial expression and a bilateral ptosis. She complained that "her neck felt loose," and she had little control over it. There was nystagmus; no other cranial nerves were involved. Distention was troublesome. The fetal heart was still heard.

February 11: The patient's condition was similar to that of the day before; she was more stuporous, but could be aroused. The temperature was around 103 degrees.

February 12: The patient was becoming progressively worse, but could still be aroused to conversation. The voice was hoarse, and there was some difficulty in swallowing. The tongue deviated to the left. She was very somnolent. The temperature was between 104 and 105 degrees.

February 13: The patient died.

This case is of unusual interest and is without doubt the most important in the series. This patient, with a definite history of influenza, developed after defervescence from a pneumonia the syndrome of descending radiculitis, beginning in the highest cervical segment, and progressing downward in the usual fashion as far only as the abdomen. Then followed an oculomotor disturbance and typical symptoms and signs of an epidemic encephalitis with fatal termination. The linking together of the influenza, radiculitis and encephalitis in this one patient suggests a helpful if not conclusive clue as to their relationship.

CASE 7.—H. P., a woman, aged 38, married, a clerk, was seen on Feb. 12, 1920.

Family and Past History.—This contained no diseases bearing on the present condition.

Present Illness.—The onset was sudden. While at work on January 12 she developed a cold in the head, with coryza, headache and slight fever. The pains in the head were localized occipitally and became rapidly worse, radiating to the base of the skull and neck. She came home, drank considerable whisky and took quinin. The pain increased in intensity and spread to the shoulders and down the arms to the hands and fingers. The pain was intermittent and lancinating in type, gradually progressing downward, radiating around the chest and abdomen and then to the groin. Here the process stopped and the pain spread more or less upward again, the pain in the occiput reappearing. There was some paresthesia and a hyperesthesia accompanied the progress of the pain. Twitching movements of the muscles were not a feature, but were noted. Delirium was not marked, but was present to a mild degree, accompanied by some confusion. Diplopia was complained of one week after the onset of her illness.

Physical Examination.—This was made by her physician ten days after the onset. The patient appeared to have received a considerable amount of opiates; she was confused and mildly delirious, the pain at times causing her to jump out of her chair. The expression of the face was mask-like. Slight ptosis of right lid and external strabismus in right eye were present. Pupils: The right was smaller than the left and reacted sluggishly to light. Movement of the neck was somewhat painful; the patient held it forward. Heart, lungs and abdomen were negative. The temperature was 101 F., pulse 100. The blood pressure was: systolic, 130; diastolic, 75. The deep reflexes in the extremities were exaggerated, suggestive of the Kernig sign. There was slight hyperesthesia to pin prick of the glove type.

Course of Disease.—The course from then on until about February 1 was a gradual abatement of the symptoms, the temperature ranging from 100 to 102 degrees, reaching a normal level one week before I saw her. Pains became

less and less frequent and severe, and the hyperesthesia abated gradually until about February 1 it suddenly became more severe, involving the entire body from neck to groin, being an intense burning pain, so that the rubbing of her clothes was almost unbearable. Asthenia had been marked, and a progressive weakness of the muscles of the arms and trunk had been noted. She had slight pain in her shoulders, chest and groin. Diplopia was still present. Physical examination on February 12 revealed a lack of emotional expression in her face; all movements were slow; slight ptosis; pupils sluggish in reaction, and an external strabismus. There was no deviation of the tongue or facial palsies. Movement of the head on the neck caused slight pain. There was no Romberg sign or gait disturbance. There was atrophy of all the spinal muscles above the waist, sternomastoids, trapezius, muscles of arm, forearm, and hand, and of the chest and abdomen, with corresponding weakness. Reflexes: Triceps, biceps, wrist jerks, knee and ankle jerks were exaggerated. No Klippel Weil. Abdominals present. There were no clonus or extensor responses. There was an occasional twitching of the muscles and a coarse tremor of the arms.

Sensorium: Above Poupart's ligament and below the thyroid cartilage over both anterior and posterior surfaces of the body there was an intense superficial hyperethesia to all forms of sensation. This includes the segmental zones supplied by the C3 to T12. Over the arms in patchy areas was a slight diminution to deep sensation of touch (pressure) and pain. Her skin was dry and the blood pressure was low.

Laboratory Findings.—January 24: Spinal puncture revealed: cells, 84: globulin +. The Wassermann test was negative for the blood and spinal fluid.

February 2: The spinal fluid showed: diminished pressure; cells, 20; lymphocytes, 100 per cent. The globulin was faintly positive; the urine was negative; white blood cells, 11,000; polymorphonculears, 80 per cent.; lymphocytes, 20 per cent.

The progress of this patient has been slow and another report will have to be made at a later date. Improvement has not again been impeded.

There are several unusual features in this case — a typical course of a descending radiculitis progressing downward as far as the last thoracic segment. Here the process reversed itself, a diplopia developed and the symptoms ascended to the occiput with hyperesthesia as the most prominent symptom. The progressive muscular atrophy, while noted to a mild degree in Cases 1, 2 and 4, was marked in this patient. We have, therefore, to deal with a lesion extensive in area — an encephalomyeloradiculitis.

Case 8.—F. S., aged 38, an exterminator, was admitted to medical A service, St. Luke's Hospital Feb. 9, 1920.

Symptoms.—The patient complained of fever, pains in body, twitching of extremities and delirium. The history was obtained from his mother.

Past History.—This was negative.

Present Illness.—He had been suffering with a badly infected tooth for three months. On January 23, after eating four portions of charlotte russe, he had a severe vomiting attack, and also complained of soreness in the tooth, which caused the throat to be inflamed. On January 27, he first noticed sharp,

darting pains in the back of the neck behind his left ear, soon traveling to the other side and to the shoulder, and a "pins and needles" sensation in the hands. Fever of 102 degrees was noted by the physician. On January 29 he complained of diplopia and blurring vision which remained until admission to the hospital. On this day the pain was severe and knife-like down the arms and in the back of the neck, and when his mother touched his hands or arms he complained a great deal of the pain produced. This hyperesthesia also involved the shoulder and back of the neck. Numbness and tingling in the arms and hands were noticeable. A spasmodic involuntary twitching of the hands and arms developed soon after so that his arms were "always moving." Within a few days the pain progressed to the upper chest followed by girdle pains around the abdomen, which were accompanied by spasms of the recti muscles. About four days before admission pains, paresthesia, and twitching progressed downward to the legs, but the pains after the first few days were never severe and while in the legs still persisted above. On February 4 he became actively delirious, talking wildly, seeing things and jumping out of bed. This continued up to and after admission. On February 6 a ptosis of the eyes was noticed by his mother, but there was no somnolence or lethargy; in fact he suffered from insomnia. The temperature was about 102 degrees until admission to the hospital.

Physical Examination.—The patient was acutely ill; actively delirious and trying to get out of bed; subsultus; at times he seemed distinctly lethargic, and could be aroused with difficulty. Eyes: There was a lack of coordination of eye movements but no definite paresis of a muscle. Photophobia was present. The pupils were unequal, contracted and fixed to light. The fundi were normal. Twitching of abdominal muscles and of legs was still present. All reflexes were present and active. There were no sensory disturbances.

Course of Disease.—February 11: The patient was delirious and could not be aroused. The pupils were as noted above and twitchings of the muscles were still present; some subsultus.

February 12: He was unable to open his eyes, but could be aroused. Twitchings were less marked.

February 13: Some twitchings were present and there were coarse tremors of the arms. Slight catatonia was noted.

February 14: The patient was not easily aroused, and had some resistance to passive motion; parkinsonian facies were suggested by his appearance.

February 17: He replied somewhat to questions. He had a delirium of a muttering type, but was more quiet and lethargic, at times somnolent. The pupils reacted to light; the right pupil was smaller than the left.

February 26: During the past week there had been slow improvement. His mental condition was clearing and he fed himself. There was less ptosis.

The temperature during the first week after admission varied between 103 and 101 degrees. During the second week it varied between 102 and 101 degrees; in the third week it was between 101 and 100 degrees. After that gradually approached normal.

Spinal fluid examinations showed cell counts of 30, 51, 120, 46, 40 and 30, the cells all being lymphocytes. Globulin was present in excess. The colloidal gold curve was: 1111.51.510000.

The blood and spinal fluid Wassermann tests were negative,

The blood count was: red blood cells, 5,500,000; hemoglobin, 100 per cent.; the white blood cells ranged from 12,000 to 16,000, and the polymorphonuclears from 74 to 86 per cent.

This case shows the syndrome of a descending radiculitis which became attenuated in its intensity as it progressed downward, but presented characteristic symptoms in the legs in conjunction with a diplopia, mask-like face, active delirium passing into a somnolent condition, a combination of radicular, midbrain, basal ganglions, and cortical lesions all progressing toward recovery.

LITERATURE ON RADICULAR SYMPTOMS IN EPIDEMIC ENCEPHALITIS

In the hundreds of case reports with which the literature on encephalitis abounds, it is of interest to note the absence of any that correspond to the cases which have appeared this winter. An occasional patient is reported to have had vague neuritis pains, but not one has had the orderly sequence of symptoms which these have presented.

In a number of excellent case reports on the disease, Barker, Ross and Irwin [3] give one which proceeded as follows: backache, hiccough, pain around the waist with a feeling of constriction, stabbing pain down to the groin and later lancinating pains down the legs. The patient was drowsy, but had no diplopia. The pupils were sluggish. The spinal fluid revealed a pleocytosis, 14 to 48 cells, and an increase in globulin. This case was considered by them to be a meningo-radiculomyelo-encephalitis, and it progressed to recovery.

Tilney and Riley,[4] in their nine subgroups of classification of epidemic encephalitis, name a posterior poliomyelitic type and cite a case which presented lethargy, diplopia, paresthesia, pain, a zoster-like eruption over the left trigeminal distribution, and then paresthesia and intense hyperesthesia over sacral second, third and fourth segments of the right leg. The process involved the dorsal root ganglion in the sacral region and the gasserian ganglion as well as the midbrain and cerebrum.

C. DeFano [5] reports a patient who developed severe trigeminal pain in the course of an epidemic encephalitis, and Buzzard [6] one of a

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3. Barker L. F.; Cross, E. S., and Irwin, S. V.: On the Epidemic of Acute and Subacute Inflammation of the Nervous System Prevalent in the United States in 1918-19: Encephalitis, Encephalomyelitis; Polyneuritis, and Meningo-Encephalo-Myeloneuritis, Part 1, Am. J. Med Sc. **159**:157, 1920.

4. Tilney, F., and Riley, H. A.: Epidemic Encephalitis, Neurological Bull. **2**: 106 (March) 1919.

5. DeFano, C., and Ingleby, H.: Preparations for Cases of Encephalitis Lethargica, Proc. Roy. Soc. Med., Section of Pathol. **12**:42, 1919.

6. Buzzard: Encephalitis Lethargica, Proc. Roy. Soc. Med., Section of Neurol. **12**:56, 1919.

pregnant woman with onset of acute pain down the left arm followed by spasmodic twitchings of the arms, diplopia of transient character and delirium. The differential diagnosis from chorea gravidarum was difficult, but he considered it an abortive case of encephalitis.

McNalty,[7] in his report on the epidemic in England in 1918, noted the following facts: Pains were not of infrequent occurrence, either diffuse all over the body or localized to particular regions, such as the neck, back or limbs. Twitchings occurred in the face and limbs. Hyperesthesia of the skin was noted especially in the limbs and soles of the feet. He mentioned five patients who had paresthesia and "neuritis" in the arms and legs.

It can be seen from these references that involvement of the posterior roots is not a new occurrence with the epidemic this winter, but there have been as yet no reports of a descending radiculitis with delirium appearing as a form of encephalitis.

PATHOGENESIS

There is definite clinical evidence from the case reports given in this article that an involvement of the posterior spinal roots is of frequent occurrence, appearing either alone or in conjunction with signs of an encephalitis. From the pathologic reports it is difficult to evaluate the extent of the lesions, for although observations on the brain are legion and reveal that any part may be affected, those on the spinal cord are few and inconclusive. As all my case reports concerned persons who had recovered, except in one instance in which no necropsy was permitted, I have been able to draw conclusions only from two reports. Strauss and Loewe[8] have noted a round cell infiltration in the posterior root ganglions. At the Rockefeller Institute Flexner and Amoss[9] have noted a like involvement with some small hemorrhages and perivascular infiltration similar to that noted in the cerebrum. With these few pathologic reports, and from the nature of the symptoms and physical signs in this spinal form of encephalitis, it seems quite certain that the lesion is located in the posterior spinal roots and possibly in the parenchyma of the posterior root zone of the spinal cord. There is some mild meningeal inflammation, and the presence of delirium indicates an involvement of the encephalon.

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7. McNalty: British Local Government Board, Report on Encephalitis Lethargica, N. S., 1918, No. 121.

8. Strauss, I., and Loewe, L.: The Etiology of Acute Epidemic Encephalitis. Presented before the New York Pathological Society, Feb. 11, 1920.

9. Personal communication from Dr. H. L. Amoss.

CONCLUSIONS

As one begins to consider the manifestations that epidemic encephalitis has shown clinically and pathologically, it becomes clear that this disease may invade almost any portion of the nervous system. It has a predilection for the basal ganglions, nuclei of cranial nerves, and, as we see from the case reports in this paper, for the posterior roots, but other parts are not exempt from its invasion.

Acute descending radiculitis is an infection of the posterior spinal roots which may appear as a separate clinical entity and pursue a most stereotyped course, eventually ending in recovery. It may also antecede, by several weeks or days, in a more or less typical but usually attenuated course, the cerebral form of epidemic encephalitis. Assuming that a posterior root syndrome may be a prominent complication of epidemic encephalitis, such symptoms as myoclonic twitchings, hyperesthesia and radicular pains may be considered as much a part of the picture in the disease as diplopia, somnolence and cranial nerve palsies.

The etiology of this disease has required considerable study. Its similarity pathologically to poliomyelitis has been noted, but as encephalitis becomes more familiar to us, this resemblance soon diminishes, and we appreciate that it has many different manifestations. It is not within the scope of this paper to discuss the history of the disease except to note that it has appeared in a similar form coincident with previous epidemics of influenza. Its occurrence in epidemic form in the winter of 1918-1919 and again in 1919-1920, with sporadic cases occurring between the two epidemics, seems to link it closely to influenza. Efforts to determine the etiologic organism in influenza have not been too decisive, the only bacteria found with frequency having occurred in its complications, most probably symbiotic with the yet undiscovered micro-organism which causes the disease. A similar uncertainty has obtained in encephalitis, for a number of investigators have reported the presence of an organism, among them Strauss and Loewe,[8] who reported a "globoid" organism a few months ago. The question still appears to be unsettled.

Reports scattered through the literature on the previous epidemics of influenza comment on its predilection for nervous tissue, and Menninger,[10] writing of the 1918 epidemic, remarks that "the quantitative specificity of the influenza neurotoxin is confirmed by its remarkable potency and versatility; the large number and wide variety of psychic and encephalopathic lesions produced being one of the most striking neuro-psychiatric features of the disease."

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10. Menninger, K. A.: Psychoses Associated with Influenza, Arch. Neurol. & Psychiat. **2**:291 (Sept.) 1919.

In a previous section of this paper was mentioned the frequency with which pains have occurred this winter as sequelae to, or independent of, an acute influenza. These have been most commonly intercostal or around the abdomen, but have also been localized in the extremities. They have been radicular in their distribution and of a neuralgic root type, sometimes lasting with great intensity for only twenty-four hours, and again resisting all efforts at relief and persisting several weeks. Hyperesthesia over the dermatomes was present in many cases. It seems that the close relationship between these radicular pains and influenza, and their similarity to the radicular types of epidemic encephalitis may suggest a possible, but of course as yet unproved, common etiology.

The author begs to acknowledge with great pleasure and thanks the privilege of observing and reporting the material in this paper in Cases 1 and 8 to Dr. Austin W. Hollis, Case 2 to Dr. F. Warner Bishop, Case 5 to Dr. W. H. Glafke, Case 6 to Dr. Kyle Steele, and Case 7 to Dr. Waldo B. Farnum.

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ENCEPHALITIS WITH INVOLUNTARY MOVEMENTS

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The four cases herein reported show such a characteristic picture that they may be classed as belonging to a somewhat individual type of encephalitis. In fact, after two of them had been described,[1] an article[2] came to the writer's attention containing a clinical picture differing in minor particulars only from that of the cases I report.

Few cases of encephalitis with involuntary movements have been described. In those reported by Buzzard,[3] the movements seem to have differed in several interesting ways from those of my patients. He states that "all these movements cease during sleep," whereas in the three cases that I observed they were much more active during sleep. Buzzard lays no emphasis on the rhythmic character of the movements — one of the most striking features in my cases. Only in Cases 10 and 12 does he speak of movements of the shoulder girdle muscles; I was particularly impressed with the bilateral and symmetrical distribution of the involved muscles and that they were especially those of the neck and shoulder girdle. In the article by Sicard and Kudelski no mention is made of the effect of sleep on the movements or of bilaterality. They give an unfavorable prognosis; whereas the four patients I described have all recovered from the acute stage and seem to be improving with respect to the involuntary movements. Bladder disturbance has been a prominent symptom and in each case the cerebrospinal fluid has shown evidence of inflammation; Sicard and Kudelski say the sphincters are unaffected and the spinal fluid normal.

REPORT OF CASES

CASE 1.—*History.*—S. H., a boy, aged 11, entered Wesley Hospital Nov. 30, 1919. Ten days before entrance he complained of a sticking or prickling sensation in the right side of the face. Two or three days later he began to vomit. On the day of entrance fever was first determined, and about 5 a. m. the boy had a convulsion; convulsions recurred during the day.

1. Med. Clin. North America **4** (May) 1920.
2. Sicard and Kudelski: Encephalites Aigues Myoclonique, Bull. et mém. Soc. méd. d. hôp. de Par. **44**:95 (Jan. 29) 1920.
3. Encephalitis Lethargica, Proc. Royal Soc. of Med., Neurol. Sec. **12**:56 (Aug.) 1919.

When first seen at the hospital he was at times rational, and complained of pain in the right side of the face; at other times he was delirious, shrieking and yelling. When I first saw him his posture was highly characteristic of meningeal irritation. The boy was lying on his side, the head was drawn back, the knees and hips sharply flexed.

Examination.—There was no opposition to passive movements, except slight rigidity of the neck, and no Kernig's sign. The pupils were dilated, reacting slightly, if at all, to light; ocular movements were normal. The reflexes could not be obtained because of the incessant movements of the patient. He had the appearance of being toxic. There was no distinct paralysis. It was impossible to test sensation. The spinal fluid was under increased pressure and showed about 1,500 cells to the cubic millimeter.

Course of Disease.—During the following six weeks the patient continued in a more or less irrational condition; he frequently involuntarily discharged urine and feces; he had no distinct convulsions. About two weeks after entrance he had to be catheterized. During six weeks the white blood count varied between 22,000 and 16,000, the temperature between 104 and 100 F., the pulse between 150 and 110. The urine was negative. On January 12 he appeared much brighter. Notes made at that time state: "There is more or less persistent, interrupted trismus; a slight spasm in the temporals and masseters, occurring about two or three to the five second period, showing no distinct regularity. There are also frequent clonic spasms of the thoracic and abdominal muscles and the diaphragm. These spasms seem to overflow into the extremities where, however, they are not so prominent. The voice is that of a patient with well developed general paresis; it has the same monotonous, slightly nasal quality, is tremulous, in the regular paretic tempo, and the syllables are not cleanly cut, mistakes in enunciation occurring in complex words. All voluntary movements are possible. The plantar reflexes are much diminished but flexor in type. The right ankle-jerk is normal, the left is absent. The right knee-jerk is normal, the left diminished. It is impossible to demonstrate the abdominals and cremasterics because of the constant involuntary twitchings. He says that a pin prick on either leg feels like cold water. It is not felt at all on the anterior surface of the thighs. There is no Kernig sign, but slight rigidity of the neck which seems almost volitional; he holds his neck rigid, then suddenly gives up and allows the chin to flex practically to the chest. The mental confusion has entirely disappeared and he can control bowels and bladder." Lumbar puncture at this time revealed no increase in pressure; the Ross-Janes test was positive; 20 cells to the cubic millimeter.

On February 10 he felt quite well and had been up and about for a week. Notes made at this time state: "The patient does not feel any involuntary movements. On examination, however, a peculiar, complex spasm or sudden contraction of the pectorals, sternocleidomastoids and diaphragm, is seen to occur about forty to fifty times a minute. It is impossible to determine whether there is a relation between the movement and respiration. He is able, for example, to take five deep inspirations without the spasms interfering with the rhythm; they do, however, interfere tremendously with ordinary quiet breathing. Associated with the movements of the above mentioned muscles there are spasms of the latissimi dorsi, the left teres group, both biceps—more spasms of the left than of the right—and the pronator and flexor groups of the left hand. At times the adductors of the left thigh twitch with the above mentioned muscles; also there are irregular spasmodic twitchings of the recti abdominis, at times together, at other times separately. A convulsive twitch

Fig. 1 (Case 1).—The line with the large excursions represents the respiration. The more irregular line of smaller excursions represents the muscular twitch. The bottom line represents the time. The synchrony of the muscular twitch and the respiration is quite obvious. For a period of ten seconds the patient held his breath. The tremor went on with an altered rhythm.

elevates the right eyebrow and retracts the right angulus oris. This is irregular in tempo, is markedly different in intensity at different moments, and occurs about eighty times to the minute. At times the movement around the eye is more marked, at others the movement around the mouth. There is tremor of the tongue, both of the trombone type and a finer tremor along the edges. No disturbance of sensation is demonstrable. Plantar reflexes are normal, ankle and knee jerks are present, greater in the right side than in the left; the abdominals are diminished, deep reflexes in the arm are present on the right side, questionable on the left. Pupils and ocular movements are normal."

On April 15, the patient was observed during sleep. The twitch of the sternocleidomastoids, the trapezei and the pectorals was more marked than during his waking hours. Fluoroscopic examination showed a twitch of the diaphragm synchronous with the twitch of the shoulder girdle muscles.

Case 2.—*History.*—T. A., a man of 45 years, was seen with Dr. A. J. Hedgcock at Decatur, Ill., on Feb. 1, 1920. I am indebted to Dr. Hedgcock for notes on the case. With the exception of a nervous breakdown five years previously, family and personal histories were negative.

While driving on the afternoon of Jan. 18, 1920, a "terrific pain" suddenly developed in the right arm. The pain kept him awake all night, and the following day it extended across the back into the left arm and up the back of the neck.

Course and Treatment.—When first seen, January 20, the patient complained of severe pain down the right arm. His temperature was 99.5 F.; the findings otherwise were negative. On January 21 the pain involved both arms and a tendency toward myoclonia was noted. The pain at this time was so intense that a hypodermic injection was necessary. The following day there was slight delirium, the myoclonia had become intense and extended to the abdominal muscles. The delirium increased, the temperature was 100 F., the pulse 90, and respirations 20. The Wassermann reaction with blood and spinal fluid was negative. There were 6 cells to the cubic millimeter of spinal fluid; the globulin test was negative. The blood showed 10,500 white cells, of which 85 per cent. were polymorphonuclears. The urine contained a trace of albumin and a few hyaline casts. Three days later pulmonary edema developed. Oxygen was given the entire night with cardiac stimulants and at 7 the next morning the patient was much better. Respirations at this time were unequal and shallow, giving the appearance of diaphragmatic involvement.

The patient was seen by me about this time. There was slight drooping of the right eyelid and weakness of the right arm. Marked twitching of the deltoids, pectorals and abdominal muscles was present. Pupillary reflexes were normal, the tendon reflexes were diminished, the Babinski sign was absent. The spinal fluid contained 10 cells to the cubic millimeter; the Nonne reaction was faintly positive. Leukocytes had risen to 15,000, of which 88 per cent. were polymorphonuclears. The delirium was of the toxic type, strongly suggesting that of alcohol.

About a week later the patient had to be catheterized, the only time involvement of the bladder was noted.

For the next few weeks a condition of toxemia continued, twice interrupted by consolidation of the lung, once on the left side and once on the right, probably hypostatic. There was gradual lysis, the delirium faded, and the only defect remaining was a gradually improving paralysis of the right arm and the twitching of the recti abdominis.

Case 3.—*History.*—A woman, 20 years old, whose family and previous history were unimportant, Jan. 16, 1920, had a stiff neck and a severe, sharp pain

running from the left side of the neck down into the shoulders and to the heart region which in the course of a few days extended down the left side to the foot. At the same time tremor of the hands and arms developed which gradually increased in intensity until, after a few days, it entirely prevented the patient from feeding herself or even raising a glass to her lips. During this time neither headache, rigors, chilliness nor flushes indicated fever. Shortly after the onset she began to have what were described as "frightened spells," with screaming. These spells occurred with considerable frequency, more often at night and apparently more often when, for any reason, she was emotionally aroused. There was also much delirious talking, especially at night.

There had been no difficulty with vision and no disturbance of speech. For three days previous to examination the urine had been hard to start, even when the desire was great.

Fig. 2 (Case 3).—The upper line charts the respiration, the middle the time marker and the lower the tremor. A definite relationship seems to exist between the twitching and respiration; there are two of the former to each one of the latter. A comparison of the respiratory tracings in Figures 1 and 2 is interesting, in that Figure 1 shows a single wave with an unbroken summit, whereas Figure 2 shows a double summit with a deep valley between. This valley was probably caused by the muscular relaxation occurring between the two twitchings. In both these cases the diaphragm was seen to twitch synchronously with the main twitching of the skeletal muscles.

Examination.—The patient was first seen on Feb. 23, 1920, at which time the following notes were made: "The patient is a thin, sallow girl, with a peculiar rash over the shoulders, chest and buttocks. She is in a profuse perspiration and obviously much disturbed by the trip she has just made to the hospital. The rash is petechial in appearance and yet on areas of the skin where there are hairs it seems to be a fine folliculitis. This impression is supported by the fact that her skin shows evidence of neglect.

There is frequent, fairly rhythmic twitching of the adductors of the arms; with each twitch there is an inspiratory movement which seems to be due to movement of the ribs. With this movement the recti abdominis are stretched upward; the umbilicus moving upward about an inch; there is no apparent movement of the diaphragm. Synchronous with this spasmodic twitching there is a twitching of the muscles of the left leg, especially the quadriceps femoris and the extensors of the toes. There is also a slight contraction of the flexors of the forearm and the pronators that occurs simultaneously with the twitching of the shoulder girdle.

Ocular movements and pupils are normal, the right optic disk is redder than the left. The left side of the face moves a trifle more than the right on volition and in emotional expression. There is no resistance to passive movements except slight rigidity of the neck.

The biceps jerks are normal, triceps and wrist jerks are absent, the right abdominal reflex is normal, the left absent. The presence of knee jerks and ankle jerks is questionable; probably those on the left are absent, and those on the right much diminished. The plantar reflex on the right is normal; on the left it is either absent or slightly in extension. The patient states that pin prick does not feel nearly as sharp on the left side, especially the left leg, as on the right. The ethyl chlorid spray is felt as distinctly colder on the right side. It is difficult to demonstrate any change in heat sensation. Joint and tendon sense apparently is normal. Roughly examined, the visual fields are normal.

With the major or chief twitch or spasm involving the respiratory muscles and, to a less extent, those of the arm and leg, is an almost constant tremor. This tremor is especially marked with active movements which, however, diminish the intensity of the chief twitch. During ordinary quiet respiration the twitch, occurring as it does about fifty times to the minute, seems to be the only respiratory act. However, when forced and deep respiration is attempted the flow of air is steady, it is not interrupted, and the muscular twitch, as for example that of the pectorals, is distinctly diminished. Ordinarily quiet breathing is superficial, determined by the spasmodic twitch of the respiratory muscles. The involuntary movements are all increased during sleep.

The patient is well oriented and her memory is good. As soon as allowed, however, she relapses into a more or less delirious condition in which she babbles unintelligibly and makes purposeless movements with the hands.

During the examination she had a "frightened spell." A look of terror and suffering appeared on her face, and she cried out "Oh, my leg, my leg, it's so cold." There were no special changes in the eyes, the body began to twitch rapidly; the left leg was partly flexed and held more or less rigid; there was a tendency to assume a position similar to the opisthotonus of hysteria. She reached for her mother, who was out of reach, then for the examiner. Respiration was rapid and a profuse perspiration appeared which soaked the sheet. The entire spell lasted about forty-five seconds without loss of consciousness.

White counts on the 24th and 25th were 14,700 and 17,000, respectively. The temperature ranged between 99.6 and 100.4 F., per rectum.

Pressure of spinal fluid was not increased; serum-albumin was increased; the Ross-Jones reaction was positive; there was no increase of cells.

Case 4.—*History.*—A man, 48 years of age, was seen with Dr. Magnuson at Wesley Hospital, March 18, 1920. About Jan. 1, 1920, while at work in a coal mine he had been struck on the top of the head by a rock, about the size of a fist, falling two or three feet. He was not knocked down, but a swelling about

half the size of a hen's egg appeared on the top of his head.[4] He worked on throughout the day and for the next three days. On the night of the fourth day a severe lancinating pain was felt just to the left of the midvertex. It woke him out of a sound sleep, and he was awake the rest of the night. The pain prevented sleep for the next three days and he thought it "would drive him crazy." At the end of this time he became amnesic; at least, he said that for three days he suffered pain and then lost consciousness. He knew nothing of the next three weeks, two of which were spent in a hospital, the last at home. He had been told that he acted naturally most of the time, but occasionally would talk "funny." He was in bed until March 5 because of weakness and giddiness.

At the time of entrance to the hospital he complained of weakness, lightheadedness and occasional throbbing pain through the stomach toward the left side of the abdomen.

Examination.—The patient was a large man with good color, but apparently far below his ordinary weight. The expression was rather "anxious and strained." He walked with the feet well apart, his steps were short and careful; there was no distinct ataxia either with open or closed eyes. All voluntary movements were well executed. The cranial nerves (including the optic disks) were normal; the reflexes were normal except that the cremasteric and abdominal reflexes appeared to be a little diminished; sensation was normal. There was frequent twitching of the sternocleidomastoids, cervical erector spinae and both trapezei—the right twitched more than the left. The twitch occurred about once per second. It was practically unaffected by voluntary movements, although when the muscles were actively used the twitch appeared to occur as an epiphenomenon. The patient was unconscious of the occurrence of this movement. Later when observed in sleep the twitching was obviously more active than during the waking period. The urine and blood were normal. The pulse varied between 100 and 80 during the nine days in the hospital. Lumbar puncture was not done. Fluoroscopic examination failed to show any twitching of the diaphragm.

SYMPTOMS IN AUTHOR'S CASES

A few of the details of these cases seem to deserve some additional comment.

Pain.—This was a prominent symptom. In all four cases it was the first symptom and was extremely severe and sharp. In Case 1 it was sufficiently intense to cause the boy to scream in the midst of his delirium. The fourth patient thought it drove him out of his mind into three weeks of delirium. In Case 3 the pain spread from the left shoulder and neck down the left side into the leg. During the one "frightened spell" observed it was complained of, especially in the leg. In Cases 1 and 4 the pain was in the right and left halves of the head,

4. Relative to the relation of the trauma to the development of encephalitis, Cleland and Campbell: The Australian Epidemic of Acute Encephalitis, J. Nerv. & Ment. Dis. **51**:143 (Feb.) 1920, assumed that head trauma may act as an agent occasioning the localization of the inflammatory process in the brain.

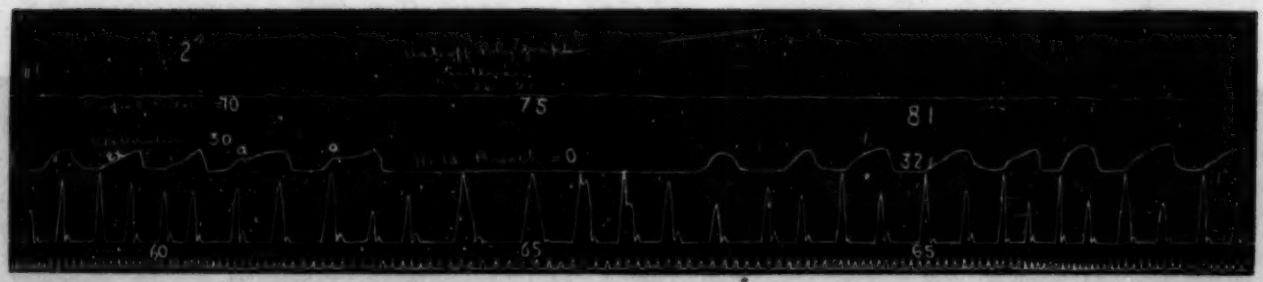

Fig. 3 (Case 4).—The upper line represents the radial pulse; the second line, respiration; the upward stroke, expiration, and the downward stroke, inspiration (see Ex. and In.). The third line represents the twitching, and the bottom line marks the time in seconds. There is a definite ratio of two twitchings to one respiratory cycle; also a slight pause (*a*) of the respiratory lever is recorded occurring midway between the summits of the regular respiratory waves. This was caused by the intervening twitching. The summits are inverted; the twitching of the sternocleidomastoid and the trapezei was sufficient to halt, momentarily, the sinking of the chest walls. There is no fixed relation between the pulse and the twitching.

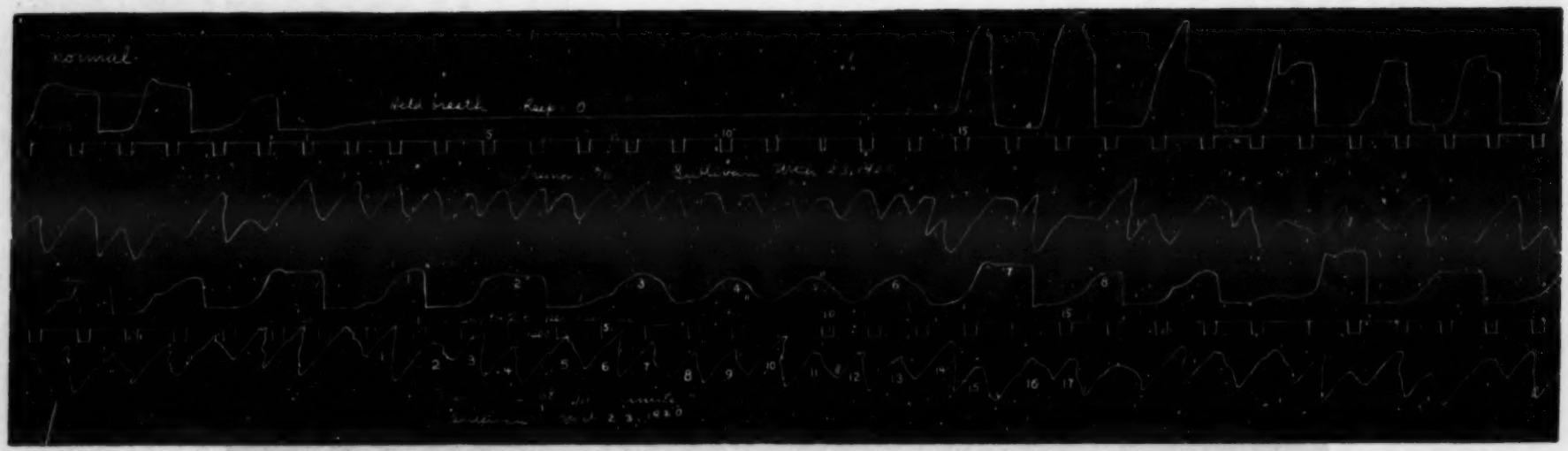

Fig. 4 (Case 4).—The top line represents respiration, the second, time; the third, the tremor; the fourth, respiration; the fifth, time; the sixth, the tremor. In this tracing there is no obvious relation between the twitching and respiration. In this case the muscular movement was much less than in either of the others, and occurred in a man of about 50 years. The apparent regularity of the tremor during the time the patient held his breath (in the upper tracing) is probably due to the fact that the respiration ordinarily moved the lever in an irregular manner. The same number of twitchings are recorded in the ten second period during which he held his breath as during ten seconds of breathing.

respectively; in Case 2 it was first in one shoulder and then in the other. Possibly this pain was due to disease of pain centers in the thalamus, as its character was suggestive of such an origin.

Fever.—In each case fever seemed to be absent for the first two or three days; at least there were no chills, rigors or flushings. The 11-year-old patient developed a temperature of 104 F.; the temperature in the other patients ranged from 100 to 101 F.

Delirium.—The mental symptoms in the three cases closely observed were a toxic delirium with restlessness, an elusive consciousness, which could be held for a few moments at a time when questions were grasped and correctly answered, hallucinations of sight and sound, and in two cases reactions strongly suggestive of terror.

Pulse and Respiration.—In Cases 1 and 3 the rapidity of pulse and respiration was out of proportion to the fever. This disproportion seemed sufficiently definite to be of diagnostic value.

Sweating.—This was especially profuse in Case 3 during the freightened spell. In Case 1 severe sweating was noticed on several occasions.

Bladder Disturbance.—This was present in three of the cases. The symptom has occurred so frequently in cases of encephalitis of this and other types that it is probably of the same nature and origin as the bladder disturbances of pseudobulbar paralysis.

Meningeal Irritation and Spinal Fluids.—In Cases 1 and 3 there was evidence of irritation of the meninges. In three of the cases there were inflammatory changes in the fluid.

Rhythmic Movements.—In the three well observed cases, the patient was quite unconscious of these movements. In Cases 1 and 3 they strongly suggested the movements caused by interruptions of a fairly strong galvanic current. It seemed incredible that the patients were unconscious of them; that they were not continually greatly plagued by them.

In Cases 1, 3 and 4 the movements were decidedly more marked during sleep. Excitement also increased them. Voluntary movement had almost no effect on them. During deep voluntary inspiration the twitches of the sternocleidomastoids could be seen going on in their regular rhythm (see tracings).

From the tracings and the clinical observations, it would appear that the clonic or twitch-like, rhythmic, bilateral movements of muscles used in forced respiration had a definite relation to the respiratory rhythm. The rate of twitching was 54 per second in Case 1, 84 in Case 3 and 60 in Case 4. That would seem too wide a variance if it were not for

the fact that the respirations varied from 54 in Case 1 to 42 in Case 2 and 30 in Case 4. In short, the rate of twitching equaled the respiratory rate or was double this rate.

Therefore it seems safe to assume that the twitches are expressions of the involvement of some complex or center in the midbrain or hindbrain that is in direct connection with the respiratory center. An arrangement controlling the muscular phenomena of respiration similar to that controlling walking may be assumed. Thiele [5] concluded from his experiments on dogs that there was a center in the posterior part of each optic thalamus that controlled the coordinated movements of walking.

If it be assumed that walking is controlled from the lower centers in a coordinated manner because of its automatic or habitual character, it seems quite likely that respiration is regulated at as low or even a lower level.

These bilateral movements can scarcely be considered as being of the nature of jacksonian cortical movements. They are too uniformly bilateral and symmetrical. It is highly questionable that cortical movements are ever rhythmic, whereas those movements that, because of their involuntary nature, suggest a subcortical control, are rhythmic.

Stewart and Pike have shown the essentially rhythmic action of respiratory centers in the medulla.[6]

I, therefore, assume that the rhythmical movements in these cases are caused by involvement of a mechanism or center in the medulla.

I am indebted to Dr. F. C. Becht of Northwestern University Medical School for a great deal of patience and persistence in securing tracings from Cases 1, 3 and 4.

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5. Thiele: Efferent Relationship of the Optic Thalamus and Deiter's Nucleus, J. Physiol. **32**:358, 1905.

6. Stewart: Some Observations on the Behavior of the Automatic Respiratory and Cardiac Mechanisms, Am. J. Physiol. **20**:407, 1907-1908. Stewart and Pike: Further Observations on the Resuscitation of the Respiratory Nervous Mechanism, Am. J. Physiol. **20**:61, 1907-1908.

DIFFERENTIAL DIAGNOSTIC DIFFICULTIES IN PERNICIOUS ANEMIA WITH ASSOCIATED MENTAL DISORDER

REPORT OF A CASE DIAGNOSED FOR SOME TIME AS ARTERIOSCLEROTIC INSANITY *

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Much has been written on pernicious anemia, particularly from the point of view of the internist and from that of the neuropathologist, but with the exception of Barrett's [1] excellent articles and Lurie's [2] more recent communication, relatively little has been contributed in English, at least on the psychiatric aspect of this condition. This is especially true with reference to the difficulties that arise in patients who present arteriosclerotic symptoms with mental features before it can be definitely demonstrated, by neurologic examination or by the blood picture, that the case is either purely one of pernicious anemia with mental symptoms, or a combination of arteriosclerosis and pernicious anemia accompanied by mental disorder. As monographic literature points out, and as hospital experience everywhere daily confirms, pernicious anemia with cord changes is more common than is recognized by the conventional textbooks on nervous and mental diseases. If the cases in general hospitals and in private practice were, or could be, followed closely by the neuropsychiatrist, a greater number of patients showing varying degrees of abnormal psychic reactions would probably be found. This might also be said of arteriosclerosis, and is borne out by the author's observation of cases seen in several outpatient services during the past three years.

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* Read before the March meeting of the Detroit Neurological Society, March 11, 1920.

1. Barrett, A. M.: Mental Disorders and Cerebral Lesions Associated with Pernicious Anemia, Am. J. Insanity **69**:1063, 1913; Mental Disorders Associated with Pernicious Anemia, Fifth Biennial Rept. State Psychopathic Hospital, Ann Arbor, Mich., biennial period ending June 30, 1916.

2. Lurie, L. A.: Pernicious Anemia with Mental Symptoms, Arch. Neurol. & Psychiat. **2**:67 (July) 1919.

There are probably many more cases of both arteriosclerosis and pernicious anemia that show associated mental symptoms than is generally recognized. Since the associated mental symptoms are not necessarily characteristic of either of these two diseases, and the blood picture characteristic of pernicious anemia in general, and the neurologic picture in particular — when the cord is involved — may both suddenly appear in cases that have hitherto shown only symptoms that belong to, or might be interpreted as peculiar to, arteriosclerosis, it becomes apparent how important it is to be guarded in any attempt to differentiate arteriosclerotic insanity from pernicious anemia associated with mental disease. It is quite possible that both conditions may exist in a given case. The following case is reported as an example.

REPORT OF A CASE

History.—(Case No. 2586, State Psychopathic Hospital, Ann Arbor, Mich.; courtesy of Dr. A. M. Barrett, director.) S. L. G., a woman, aged 54, married, housewife, an American, was one of nine children. One sister died of pneumonia, and one of cancer, ages not given. One brother died of cerebrospinal meningitis at the age of 16, and another brother who was feebleminded, died at Traverse City (Michigan State Hospital). The family history was reported as otherwise negative.

The patient was bright in school, but somewhat retiring and seclusive in her habits, preferring books and home to outside companionship. She married, happily, at the age of 20. She was a good housewife, but inclined to be overeconomical, disinclined to have a dollar spent unless it was absolutely necessary. Her religious life was not out of the ordinary.

Previous Medical History.—This was reported as negative, except for a brief "nervous spell" twelve years before.

Present Illness.—According to the husband, the present illness was gradual in onset, and extended over a period of at least three years previous to admission to the State Psychopathic Hospital on May 27, 1919. At that time the patient expressed the desire to take care of the chickens in order that she might have the proceeds from that part of the farm business. This idea apparently grew out of the patient's desire to raise sufficient funds to visit a son who was then living in the West. The husband granted her request and agreed to do the more difficult work, but insisted that she look after the poultry. She did so for a year with fair success. Then (two years ago) she began to develop delusions of a whimsical character. Because the hens were laying poorly and the chickens dying off, she imagined her husband was killing them and selling them and the eggs in order to buy dresses and furniture for a woman close by. Later she included every woman of the neighborhood in her delusions but never set traps to catch or expose her husband. Then she imagined everybody was ridiculing her. She began to suffer from sleeplessness and restlessness and lost weight. A year before admission, she began to have hallucinations and grew irritable, and on several occasions threatened her husband. She frequently showed emotional instability, crying most of the time. The husband furnished no history of any similar previous attacks, but spoke vaguely of her suffering a "nervous breakdown" about twelve years

before, being confined to bed for about three weeks. After recovering she worried about things in general and did not seem to reach her normal level for almost a year.

Physical Examination.—On admission, the patient appeared to be in a generally rundown condition. The musculature was poorly developed and of poor tone. There was almost no panniculus. The facial expression was one of apprehensiveness. The skin was dry, warm and fairly elastic, but showed a marked pallor. There were no scars, eruptions, or abnormal pigmentations. The neck was negative except for noticeable venous pulsations. The tongue showed only a slight fine tremor. Examination of the cardiovascular system revealed that the apex of the heart was in the fifth interspace at the nipple line. The heart sounds were fairly well heard and the only abnormality was a slight systolic murmur at the apex which was apparently hemic in type. The arteries and veins showed slight, if any, evidence of sclerosis, but there was some varicosity of the veins in the lower extremities. The pulse (rate 100) was of fair quality, rhythmic and synchronous. The blood pressure was systolic, 150; diastolic, 96. A week later, the heart sounds were of poor quality, accentuated in all areas, and the blood pressure was systolic, 160; diastolic, 100. There was a distinct arcus senilis in each eye.

Neurologic Examination.—On admission the neurologic examination was negative except for fine tremors of the extended hands, tongue and closed lids.

Laboratory Examinations.—Urinalysis showed nothing of any significance with the exception of a rather low specific gravity (1.002) and the presence of a few white blood cells.

Blood: Examination of the blood showed a red count of 4,000,000 and a white count of 5,850, a hemoglobin of 75 per cent. and a negative Wassermann test.

Mental Examination.—There was nothing unusual in the patient's general appearance and attitude toward the examiner except for a slight flushing of the face and an inclination to be playfully cynical. At times during her narrative she was slightly emotional. There was no evidence at that time of any abnormal psychomotor activity other than that which appeared to be associated with the slight circulatory disturbance, mild tremors and arrhythmia with inconstant tachycardia.

She denied dreaming, but admitted that she had frequent crying spells which prevented her from going to sleep. Memory for remote events was unimpaired. Simple calculations were done deliberately and slowly. She returned the "Shark Story," however, as follows: "The son of an oriental governor was on a boat when he was struck by a shark. He was cast overboard."

The patient was correctly oriented in all three spheres. Her stream of thought was clear, but not always relevant, and was very circumstantial. She went into endless details about her husband's mistreatment of her in the matter of the chickens, and it was almost impossible to keep her from reverting again and again to this topic. Except for this delusional trend she was logical.

The patient's content of thought has already been referred to in the anamnesis furnishel by her husband and in the preceding paragraph. It is further elaborated in the patient's own narrative, which adds facts of value relative to her physical condition as well as to the nature of her mental disturbance. In addition to the "nervous" condition (twelve years before) mentioned by her husband, she spoke of having had some form of cardiac disturbance four

years before admission. She fell to the floor and was unconscious for an hour. A year later she had a similar attack. Both attacks were sudden in onset, and were preceded by a feeling of weakness but not accompanied by vertigo. The account of these attacks was confirmed by her husband. Her mental symptoms developed about that time.

Her narrative, condensed, was as follows: "I was anxious to see my son who was in the West. My husband, feeling that we could not bear the expense of the trip, led me to ask him to turn over the profits of the chickens to me. Mother gave me some chickens as personal property, he took the money from the eggs and pretended to pay the grocery bills with it. When I complained of having no clothes he said I could have all the profit from the chickens. That was three years ago." She then went into endless detail about his deceit, attention to other women, etc., and continued: "About a year ago this grew gradually worse. I became nervous, cried a great deal, and was almost frantic over my losses. Last Christmas he even went so far as to change a rooster on me. One afternoon he took me to town; on our return eleven pullets were gone. Later in January, I received a message from my son in the West that he had influenza. I went to town for the telegram and on my return found more chickens changed and gone. The longer it went on the crazier I became over it."

When questioned closely why her husband did all these things she could offer no other reason than that he was not contented with her. She apparently had no insight into her condition. That her husband had been considerate and patient with her was confirmed later by the son from the West and by neighbors.

Ophthalmologic Examination.—On June 5, nine days after admission, the patient was referred to the Department of Ophthalmology for an opinion relative to the condition of the fundi. They reported, in part:

Right: Arteries somewhat contracted; some arteriovenous compression; nerve head pale and yellow in color; very heavy choroidal ring; some of the veins slightly tortuous. Foveal reflex not made out.

Left Eye: Lamina cribrosa veiled. Both the arteries and the veins somewhat contracted.

Diagnosis: Anemic fundus; angiosclerosis.

Heart Examination.—She was referred to the department of internal medicine for an examination relative to the condition of the heart. This report was made: Soft systolic at the apex—aortic roughening—possibly hemic murmur; no evidence of decompensation.

The last report was dated July 1, thirty-five days after admission. Up to that time there had been no change in the patient's general physical or neurologic condition, but the mental symptoms had become more acute, although this change was gradual, with variable intervals of relative quiet. On June 9, she insisted, and for many days repeated the idea, that she had been sent to the hospital to be a "subject," to be "ground up in a machine," and was certain that "sanitariums have to have subjects." She became restless, was frequently out of bed, refused food, saying that she could not digest it, and had periods of alternating talkativeness and uncommunicativeness. For the next two or three weeks she repeated her fears of being "ground up" almost daily, but never with a convincing air. At night, however, she occasionally paced the floor of her room and begged the nurse or the physician to put her out of the way and end her agony.

Diagnosis.—On June 25 her case was presented before a staff conference, with the diagnosis of arteriosclerotic insanity. This diagnosis was accepted by those present at the conference, and the case was entered under this classification on the hospital records. The diagnosis was reached on the basis of this summary of the case:

1. Family History: This was negative except for one feeble-minded or insane brother.

2. Gradual Onset: The onset was at least three or four years in duration.

3. Symptoms and Episodes Before Admission to the Hospital: Three or four years ago she had mild apopletic or syncopal attacks, two years ago she was sleepless, restless, lost weight, and had mental symptoms, chiefly delusions of persecution of a whimsical character and ideas of infidelity. Finally she became irritable and threatening and emotional instability was quite marked.

4. Physical Findings: There were malnutrition, pallor of the skin, flabby musculature; cardiovascular symptoms, including a faint systolic murmur at the apex. The blood pressure was systolic, 160; diastolic, 100 She had arcus senilis, anemic and angiosclerotic fundi and mild varicosities.

Laboratory Findings: These and the serologic findings were negative.

Mental Symptoms: There were emotional instability; slight disturbance of memory for recent events; circumstantial and at times, irrelevant stream of thought; delusions of persecution by her husband and of infidelity (never firmly voiced by the patient). These were the leading symptoms during the first three weeks' residence in the hospital. Gradually the mental symptoms became more severe and included terrifying delusions of bodily torture, apprehensiveness, agitation, increased psychomotor activity and transient periods of mild confusion.

Change in the Picture.—About July 1 she was again referred to the Departments of Ophthalmology and Internal Medicine. The former reported "angiosclerosis in both fundi with some appearance of anemia"; the latter reported "systolic murmur and symptoms of simple anemia, advised simple tonics." On July 3 another blood count was taken which showed the following startling results: 1,420,000 red cells; 1,530 white cells; differential count—small lymphocytes, 20 per cent.; large lymphocytes, 24 per cent.; transitionals, 9 per cent.; polymorphonuclear neutrophils, 45 per cent.; eosinophils, 2 per cent.

On the same day that this count was made, another neurologic examination revealed the following positive findings not noted in repeated previous examinations: The biceps and triceps reflexes were increased on both sides. The knee jerks and Achilles' jerks were not obtained on either side. Abdominal and epigastric reflexes were not obtained. When the tuning-fork was applied to the bony prominences, there was no perception of vibration from the patellae down, including both tibial and malleolar surfaces. Both conjunctivae were hypesthetic, the left more so than the right. The blood pressure had fallen to systolic, 102; diastolic, 60; but the pulse rate was 102. The right fundus showed what appeared to be small hemorrhagic foci. The case was again referred to the Department of Ophthalmology, which replied, on July 8:

Right Eye: Media clear. Physiologic disk plus. Lamina cribrosa veiled; nerve head oval; long axis vertical. Rings plus. Choroidal conspicuous in whole lower half of disk. Arteries somewhat contracted and pale. Some arteriovenous congestion. Veins somewhat tortuous, although no engorgement. Just below the disk, down and nasally about one third of a disk diameter, is a small round hemorrhage, petechial state, recent in part, the

other portion old and has been absorbed. Along the temporal veins about the disk down, are absorbed hemorrhages, below the retina. Foveal reflex not made out.

Left Eye: Vessels essentially same as in right eye. Just one-half disk diameter below the disk along the vessels are two grayish-white spots apparently old hemorrhages. No venous hemorrhages seen in this eye. Foveal reflex not made out. Edema of the retina in both eyes.

Another blood count made four days later, July 7, showed: red cells, 1,240,000; white cells, 1,600; small lymphocytes, 20 per cent.; large lymphocytes, 30 per cent.; transitionals, 4 per cent.; polymorphonuclear neutrophils, 30 per cent.; eosinophils, 2 per cent.; normoblasts, 4 per cent.; megaloblasts, 10 per cent. There were many stippled cells and oval-shaped red cells; polychromatophilic red cells were also observed. The blood count did not change for the better, the red cells falling as low as 900,000, and the hemoglobin ranged between 30 per cent. and 50 per cent.

The mental condition became progressively worse. She became garrulous and rambling in conversation. At times she was unclear and disoriented, and became fatigued easily on the least mental effort. Terrifying delusions became more pronounced; she was to be "ground up" in terrible machines; her arms, hands and legs were to be "cut off," etc. Her mood and manner, however, were frequently out of harmony with such depressing delusions; she was cynical, playful and jested in a gruesome way. She had temporary flareups of slight improvement parallel with a slightly higher red count; but on the whole she grew steadily worse. She talked of receiving electric baths at night and of poison in her food and drink. On August 6 she became uncontrollable, had to be isolated, and died on August 19. The family refused necropsy.

Change of Diagnosis.—It is hardly necessary to state that the diagnosis was changed from that of a case of simple arteriosclerotic insanity to one of pernicious anemia with associated mental disorder. The arteriosclerotic element could not be ignored, however, and the case taken as a whole appeared, from the clinical standpoint, to be one of pernicious anemia with marked mental symptoms, particularly paranoid tendencies in a person showing arteriosclerotic symptoms.

This case shows the difficulty of differential diagnosis in pernicious anemia. The arteriosclerotic symptoms appeared first and obscured the final diagnosis. Added to this, the mental symptoms, when the case was first seen, were such as could occur in either pernicious anemia, arteriosclerosis or, as Barrett has pointed out, in other mental disorders, symptomatic of toxic conditions.

LITERATURE ON PERNICIOUS ANEMIA WITH ASSOCIATED MENTAL DISORDERS

At this point, it might be well to review briefly the literature on pernicious anemia with associated mental disorders. Until the appearance of Barrett's papers,[1] this phase of the disease had received little consideration in the literature. The mental disturbances noted in this

condition had apparently been considered an incidental, unorganized terminal delirium, no dominant or characteristic psychosis having been noted.

Barrett, in his series of eleven cases, was able to determine in six of these a more or less characteristic psychopathic process, marked essentially by irritability and a definite paranoid trend, the exact nature of the specific delusions depending essentially on the particular syndrome and environment. Auditory hallucinations were observed in some cases and in others, definite confabulation. No marked deterioration was observed, the personality apparently having been relatively well preserved. Remissions in the psychic disturbance were frequently found to accompany remissions in the more essentially somatic aspect of the disease. The picture is essentially that of a gradually developing chronic condition, paralleling in a general way the progress of the actual somatic condition. Barrett suggests classifying these cases clinically among the paranoid conditions, symptomatic of toxicorganic processes.

Marcus [3] has noted that suggestive mental symptoms may manifest themselves for some time before it is possible to establish a diagnosis of pernicious anemia on the basis of the characteristic blood changes and other clinical findings. Langdon [4] directs attention to the same possibility and designates such cases prepernicious anemia. Initially, our case seems to have approximated this type.

Lurie,[2] reviewing the field in a recent article, seems to endorse Barrett's classification of the condition as a toxicorganic psychosis, remarking in addition, that the "whole delusional formation is vague, unsystematized and loosely connected." In an analysis of four cases, Lurie finds definite visual hallucinations in two cases and doubtful visual hallucinations in one other and delusions of persecution and paranoid ideas in all four.

DIFFICULTY OF DIFFERENTIAL DIAGNOSIS IN CASES LIKE THE AUTHOR'S

It was impossible to find any reference in the literature bearing directly on the question we discuss, i. e., the necessity and difficulty of differentiating between pernicious anemia with mental symptoms and arteriosclerotic insanity.

For this reason the present case is reported. During residence in the hospital, the case was looked on as one of arteriosclerosis until about a month from the day of admission, then, within the short period

3. Marcus, H.: Psychose bei perniciöser anaemie, Neurol. Centralbl. **22**: 453, 1903.

4. Langdon, F. W.: Nervous and Mental Manifestations of Pre-Pernicious Anemia, J. A. M. A. **45**:1635 (Nov. 25) 1905.

of three or four days, the whole picture became dominantly that of pernicious anemia associated with cord symptoms and mental disorder. This called for a review or what might be called an inventory of the early history of the case as well as of the symptoms up to the time when the picture became unmistakably that of pernicious anemia. Features that had been looked on as primarily arteriosclerotic, now were just as easily interpreted as those of the so-called "prepernicious" phase of the case as a whole. These features were:

1. Tainted heredity (Barrett).

2. Gradual onset, three or four years, possibly twelve, with symptoms, especially syncope, etc., which inaccurately reported by members of the family may well have been those due to anemia as well as to arteriosclerosis. Had a blood count been taken in the earlier attacks, the interpretation of the history might have been different.

3. Delusions of a whimsical character and delusions of persecution (not necessarily characteristic of arteriosclerosis).

4. Remissions in which the patient appeared to have recovered. These symptoms running back over a period of twelve years might be only "prepernicious" symptoms. Then appeared the findings and symptoms that clouded the diagnosis; cardiovascular symptoms, including soft systolic murmur at the apex, blood pressure of 160 to 100, arcus senilis and angiosclerotic fundi.

Then occurred rapid changes certainly indicative of a case of genuine pernicious anemia: low red cell count, characteristic differential count, stippled cells, etc.; cord changes, including lost patellar reflexes and lost vibratory sense, and finally the rapid and progressive aggravation of all the mental symptoms.

In conclusion, we wish to emphasize these points:

1. Probably more cases of pernicious anemia are mistaken for arteriosclerosis than are generally recognized.

2. Patients with doubtful cases of arteriosclerosis, especially those with a history of remissions, should, as a routine procedure, have frequent blood counts made, and neurologic examinations for cord changes.

3. Careful history taking may reveal the so-called "prepernicious" symptoms in cases that otherwise might be mistaken for arteriosclerosis.

4. The lack of comment in the literature on this phase of neuropsychiatry is significant, and shows that the necessity of differentiating carefully in doubtful cases has not yet been fully recognized.

OBSERVATIONS ON THE PATH OF FARADIC SENSIBILITY*

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The present study deals with the afferent path of faradic stimuli. The evidence here presented has been largely gathered from a study of cases presenting certain neurologic lesions or psychiatric conditions, and suggests that faradic stimuli of liminal value probably follow the path of the thermal fibers, while markedly supraliminal stimuli may use other paths. As was to be expected, the postrolandic cortex is in all likelihood the arrival platform for these impulses.

METHOD USED IN MAKING TESTS

The method used eliminates subjective influences both from the patient and operator. Heretofore, quantitative sensory tests have been of only relative accuracy and most unsatisfactory. Even the ordinary qualitative sensory tests are not perfectly objective. There have been three chief objections to previous attempts at quantitating tests of sensation: First, lack of absolute accuracy, i. e., the difficulty of reproducing exactly a stimulus of given intensity at will; second, lack of objectivity, and third, the ease of deception by the patient. These three factors of error have been eliminated by employing the method devised by Martin [1] for the measurement of shocks delivered by the secondary coil of an inductorium in absolute units which he called beta units. By the use of this method any given stimulus can be reproduced exactly. The subject has no means of knowing when the stimulus is applied, as he sits in another room, and no noise warns him of the application of the stimulus. The operator has an absolute check on the accuracy of the patient's replies, by stimulating the patient with

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*Study No. 6 in a series entitled "Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects."

*This work was done during the tenure of an internship at the Psychopathic Hospital, Boston.

1. Martin, E. G.: A Quantitative Study of Faradic Stimulation. I. The Variable Factors Involved, Am. J. Physiol. **22**:61, 1908. Ibid.: II. The Calibration of the Inductorium for Break Shocks, ibid. **22**:118, 1908; ibid.: III. The Measurement of Make Shocks, ibid. **24**:269, 1909; ibid.: IV. The Make and Break Key, ibid. **26**:181, 1910; ibid.: V. The Influence of Tissue Resistance and of Kathode Surface on Stimulating Effectiveness, ibid. **27**:226, 1910; ibid.: VI. The Comparison of One Inductorium with Another, ibid. **28**:49, 1911; ibid.: Measurement of Induction Shocks, New York, 1913; ibid.: On the Validity of Inductorium Calibrations, ibid. **36**:223, 1915.

shocks of varying intensity, any one of which may be repeated as often as is deemed desirable. A brief description of the method as employed by us follows.

The measurement of shocks delivered by the secondary coil of an inductorium by this method depends on the fact that with a given current passing through the primary the shock delivered by the secondary coil (on making or breaking the primary circuit) depends on the proximity of the secondary to the primary coil in any given plane. The exact relation between shock delivered and intercoil distance varies with the inductorium used. This must be calibrated for both make and break shocks.[2] Throughout this work only break shocks were used. These were delivered by the "Make-and-Break Key," [3] the advantage of which lies in the fact that it allows a uniform make or break as the case may be, and enables one to short-circuit the one or the other.

The shocks from the secondary were delivered to the patient by electrodes of the mercury-calomel-salt-solution type described by Martin, Porter and Nice [4] as the most advantageous for work with human subjects. They also found that abrasions of the fingers used in the test (or the use of platinum points thrust under the skin as electrodes) lowered the average human threshold markedly. This is a practical point of some importance in dealing with maniacal patients who often sustain slight injuries. These electrodes consist of two tubes about 5 inches long and of a diameter to admit the fingers comfortably. The wires from the secondary coil are led to the bottom of these tubes (one into each tube) over the top of the tube. The insulation of the wires is stripped for a distance of 1 or 2 cm. from their ends, which rest on the bottom of the tubes. Around the ends of these wires is a layer of mercury, over which lies a layer of calomel paste to a depth of 2 cm. The rest of the tube is filled with 15 per cent. salt solution.

The patient dips the middle and index fingers of his left hand into the electrodes and is instructed to press a telegraph key whenever he feels a shock. The operator starts with the secondary coil in such a position that it will deliver a subliminal shock, and gradually approaches it to the primary, breaking the current at short irregular intervals. Finally, the patient signals, indicating that he has felt the shock. This

2. Martin, E. G.: Am. J. Physiol. **22**:118, 1908; ibid. **24**:269, 1909; ibid. **28**:49, 1911; ibid. **36**:223, 1915.

3. Martin, E. G.: Am. J. Physiol. **26**:181, 1910.

4. Martin, E. G.; Porter, E. L., and Nice, L. B.: Sensory Threshold for Faradic Stimulation in Man, Psychol Rev. **20**:194, 1913.

point and its environs are tried and retried until the point farthest from the primary is found where the patient feels practically every shock. This can be found within 2 mm. as measured on a scale which has as its zero the point where the secondary lies over the primary and encloses it. This procedure is repeated three times with ten, twenty and thirty thousand ohms, respectively, introduced into the primary circuit. From this it is evident that deception on the part of the patient is impossible. The entire procedure consumes about six minutes, on the average, though perseverance will frequently procure a reading from a subject who at first seems to offer no cooperation whatever. The resistance of the coil used in this work is 2,900 ohms. In work of the highest accuracy it is necessary to determine the resistance of the patient's skin by the somewhat tedious Kohlrausch method. This resistance has been found to be very constant, however, and in our work we felt that it was better not to determine the resistance in each case, on account of the time necessary for the procedure and the restiveness of certain patients. We have taken the skin resistance to be 2,100 ohms, the average of a large number of observations, three quarters of which fall within 300 ohms of this figure. This omission introduces only a small percentage error, especially in dealing with high threshold values. Thus, in the secondary circuit there is the resistance of the secondary coil (2,900 ohms) plus the resistance of the patient's skin (2,100 ohms) or 5,000 ohms. Our four readings of the position of the secondary coil are made with a total of five, fifteen, twenty-five and thirty-five thousand ohms' resistance in the secondary circuit. From these resistances, the value of the current passing through the primary coil, and the position of the secondary coil, the value of the threshold shock is calculated by certain formulae developed by Martin [5] as modified by Wilbur.[6] The primary current used throughout this work (except in the cases of syringomyelia) was 0.3 ampere.

Throughout this work the results have been expressed in the number of beta units required to cause sensation. One beta unit is the average amount of faradic shock necessary to cause a minimal contraction of a frog's gastrocnemius stimulated through the sciatic nerve in the classic nerve-muscle preparation.[4]

It is to be emphasized that in interpreting the results here presented, the higher the figure given (in beta units) the less sensitive is the subject.

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5. Martin, E. G.: Measurement of Shocks, New York, 1913.

6. Martin, E. G.; Bigelow, G. H., and Wilbur, G. B.: Variations in the Sensory Threshold for Faradic Stimulation in Normal Human Subjects. II. The Nocturnal Variation, Am. J. Physiol. **33**:415, 1914.

PREVIOUS WORK

Since the introduction of this method in 1908, considerable work has been done on the variations of this threshold in normal and psychopathic human subjects and in the experimental animal under various conditions. Martin, Porter and Nice [4] found that there was a practically constant threshold value for a normal human subject, tested as described above. As a result of introspective observations, they suggested that this form of sensation was allied to muscle or joint sensibility. Continuing the work on normal human subjects certain evidence was accumulated to show that this method might be used as an index of the functional condition of the central nervous system. It was found that this threshold had a definite diurnal variation [7] which corresponds closely with the curve of ergographic output as reported by Lombard,[8] and with the diurnal variation of the reaction time as recorded by Ostanikow and Gran.[9] The lack of closer correspondence with the latter curve may be ascribed to the fact that these authors made only four observations in the course of a day as compared with hourly observations in the study on this threshold. Martin, Bigelow and Wilbur [6] worked out a nocturnal rhythm which was found to agree closely with the depth of sleep as measured by Mönninghoff and Piesbergen,[10] the periods of low irritability coinciding with those of greatest sleep intensity. It was further found in this study that the second low point in irritability corresponds with the point of lowest body temperature.[11] The influence of general fatigue on this threshold is of some importance in the use of the threshold value clinically for the examination of ambulatory cases. In this study [12] it was found that men under a somewhat pressing but regular routine showed a curve of decreasing irritability as the week wore on. This curve was

7. Grabfield, G. P., and Martin, E. G.: Variations in the Sensory Threshold for Faradic Stimulation in Normal Human Subjects. I, The Diurnal Rhythm, Am. J. Physiol. **31**:300, 1913.

8. Lombard, W. P.: Some of the Influences which Affect the Power of Voluntary Muscular Contractions, J. Physiol. **13**:25, 1892.

9. Ostanikow, P., and Gran, M.: Ueber die Geschwindigkeitsveränderungen der psychischen Processe zu verschiedenen Tageszeiten, Neurol. Centralbl. **12**:290, 1893.

10. Mönninghoff, O., and Piesbergen, F.: Messungen über die Tife des Schlates, Ztschr. f. Biol. **19**:114, 1883.

11. Jürgenson and Liebermeister: Handbuch der Pathologie und Therapie des Fiebers, Leipzig, 1875; quoted by Pembrey in Schäfer: Textbook of Physiology, Edinburgh and London **1**: 1890. Jürgenson, T.: Die Körperwärme des Gesunden Menschen, Leipzig, 1813.

12. Martin, E. G.; Withington, P. R., and Putnam, Jr., J. J.: Variation in the Sensory Threshold for Faradic Stimulation in Normal Human Subjects. III. The Influence of General Fatigue, Am. J. Physiol. **34**:97, 1914.

obtained by testing the men at the same time each day. With the intervention of Sunday and relief from their routine, the irritability increased to the point where it had begun the week before. Martin, Paul and Welles [13] found the threshold of the winking reflex in man to be about twice that of the sensory threshold and concluded that "the value of the sensory threshold depends on the state of the higher nervous centers, and that the threshold may justly be used, therefore, as an indicator of such nervous state."

The indication that "synaptic resistance" probably has an effect on this threshold suggested certain animal experiments which showed that in the spinal cat [14] the threshold for the radial nerve-muscle preparation is almost exactly half as high as that for the flexion reflex obtained by stimulating the tibial nerve. In a further study of the variations in the threshold of the reflex arc [15] it was found that an excess of carbon dioxid raised the limen of the flexion reflex in the spinal cat to the point of its total disappearance, and that this effect varied with the percentage of oxygen present. This suggests that a study of neuropathic cyanosis may permit of correlation with this observation.

Finally, a group of experimental studies [16] on the limen of the vasomotor center showed that the intensity of the stimulus to the nervous system may make a difference in the responses to two stimuli varying in strength and not in kind. These studies also showed that the threshold of the vasomotor center is about twice that of the flexion reflex and about four times that of the nerve-muscle preparation of the spinal cat. This may possibly be taken as further evidence of the effect of the interpellation of synapses on such tests.

At this time the work was carried over to an examination of psychopathic human subjects. In a preliminary survey [17] it was found that the threshold of the psychopathic patient tends to be high, the average

13. Martin, E. G.; Paul, B. D., and Welles, E. S.: A Comparison of Reflex Thresholds with Sensory Thresholds. The Relation of This Comparison to the Problem of Attention, Am. J. Physiol. **26**:428, 1915.

14. Porter, E. L.: Thresholds of Electric Stimulation in the Spinal Cat Determined by the Martin Method, Am. J. Physiol. **31**:141, 1912.

15. Porter, E. L.: Variations in Irritability of the Reflex Arc. I. Variations Under Asphyxial Conditions, with Blood Gas Determinations, Am. J. Physiol. **31**:223, 1913.

16. Martin, E. G., and Lacey, W. H.: Vasomotor Reflexes from Threshold Stimulation, Am. J. Physiol. **33**:212, 1914. Martin, E. G., and Stiles, P. G.: Two Types of Reflex Fall of Blood Pressure, ibid. **34**:106; 1914; ibid.: The Influence of Curare on Vasomotor Reflex Thresholds, ibid. **34**:220, 1914; ibid.: Some Characteristics of Vasomotor Reflexes, ibid. **37**:94, 1915.

17. Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: First Note, Boston M. & S. J. **161**: 883, 1914.

value in this group of cases being 223 beta units as against 100 beta units in the normal person. It was further shown that this test was a feasible one for routine use in psychopathic subjects.

The larger psychiatric entities were then studied. In the manic-depressive group [18] it was found that the maniacal patients had a normal or slightly lowered threshold, and that the depressed patients showed a faradic hypesthesia which wore off as the depression disappeared. It was felt that this variation in the threshold was probably intimately connected with the fundamental cause of the variations in emotional tone in these cases. Kraepelin [19] believes that this disease is caused by a congenital predisposition to mania or depression, as the case may be, and that the alternating forms may be found either in the manic or the depressive temperament as a basis. He further believes that this predisposition is manifested in the personality of the person when he is normal. He uses the fact that maniacal attacks may be superposed on the depressive temperament as an argument for his contention that mania and depression are different degrees of the same process and not opposites as would appear. The threshold for touch, temperature and pain has been found to be high in depressed patients as measured by tests of relative accuracy. The association time in manic cases is somewhat lengthened, but is more so in depressed cases. Southard [20] has shown that there is an absence of gross brain lesions in the brains of manic-depressive subjects, and his findings and that of others have indicated the functional character of this disease. This same observer has noted lesions of the thalamus in cases [21] other than manic depressive insanity, that showed during life hyperkinesis and its psychical accompaniments. Similarly, Bevan Lewis [22] has described an absence of metabolic pigment in hyperkinetic states, and Southard and Bond [23] have shown that there is an increase of metabolic or catabolic lipoid substances in the cortex of depressed cases of manic depressive insanity. From the work on the thalamus it was suggested

18. Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: II, Manic Depressive Insanity, Boston M. & S. J. **173**:148, 1915.

19. Kraepelin, E.: Psychiatrie, Ed. 8, Leipzig, 1913.

20. Southard, E. E.: Anatomical Findings in the Brains of Manic Depressive Subjects, Proc. Am. Psychol. Assn., May, 1914.

21. Southard, E. E.: The Association of Various Hyperkinetic Symptoms with Lesions of the Optic Thalamus, J. Nerv. & Ment. Dis. **41**:617, 1914.

22. Lewis, W. Bevan: A Textbook of Mental Diseases, Philadelphia, P. Blakiston's Son & Co., 1890.

23. Southard, E. E., and Bond, E. D.: Clinical and Anatomical Analysis of Eleven Cases of Mental Disease in the Second Decade, with Special Reference to a Certain Type of Cortical Hyperpigmentation in Manic Depressive Insanity, Am J. Insan. **71**:194, 1914-1915.

that the hyperkinetic states might be associated with a withdrawal of the corticothalamic "inhibitory" or "switch-setting" impulses, that is, with a "tissue simplification." It is possible from the histopathologic evidence in the opposite state that these depressions may be associated with an accumulation of catabolic products. In view of this evidence and the reactions we have demonstrated in the receptor mechanism, it was suggested as a working hypothesis that these two contrasted states in this psychosis might be brought about by a functional hypertonicity or hypotonicity (for depression and mania, respectively) of these or similar centers. This hypothesis is further supported by Sachs'[24] demonstration of the intimate relation between the thalamus and sensation. Additional suggestive evidence as to possible variations in the reactions of such centers is the demonstration that with threshold stimulation of a peripheral nerve the reflex vasomotor center[25] responds with a depressor effect, while with markedly supraliminal stimulation it responds with a pressor effect.[26]

In the dementia praecox group, on the contrary, no such variation with emotional tone is observable in the faradic threshold,[27] and it was concluded from this that the phenomenon of emotional tone in this group was probably governed by a different mechanism than in the manic-depressive syndrome. In the diagnosis of these cases we have largely followed Kraepelin, i. e., cases ending in deep dementia and displaying as their chief symptoms basic disorders in the personality, progressive deterioration and absence of the tendency to recover. There was found a close parallelism between cases showing symptoms of catatonia (silliness, grimacing, attitudinizing, stupor, cerea flexibilitas, negativism, etc.) and high threshold values, and between low threshold values and absence of such symptoms. These symptoms have been correlated with postrolandic lesions in these cases by Southard,[28] and this finding suggests the postcentral cortex as the "arrival-platform" of faradic sensation. Similarly, patients with somatic delu-

24. Sachs, Ernest: On the Structure and Functional Relations of the Optic Thalamus, Brain **32**:95, 1909.

25. Porter, W. T.: The Vasotonic and the Vasoreflex Center, Am. J. Physiol. **36**:418, 1915.

26. Porter, W. T.: The Relation of Afferent Impulses to the Vasomotor Center, Am. J. Physiol. **27**:276, 1910.

27. Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: III, The Dementia Praecox Group, Boston M. & S. J. **173**:202, 1915.

28. Southard, E. E.: A Study of the Dementia Praecox Group in the Light of Certain Cases Showing Anomalies or Scleroses in Particular Brain Regions, Proc. Am. Medico-Psychological Assn., May, 1910; ibid.: On the Topographical Distribution of Cortex Lesions and Anomalies in Dementia Praecox with Some Account of Their Functional Significance, Am. J. Insan. **71**:382, 1914-1915.

sions have shown higher threshold values than patients not exhibiting such delusions. This lends support to the hypothesis that these delusions are often a result of disturbances in the receptor mechanism, although they may have their basis in fact, as shown by the lesions found at necropsy in the organs about which the delusions occurred in life.[29] Thus from this group there has been obtained evidence to show that the arrival platforms for faradic sensation are probably located in the postcentral gyrus with other forms of peripheral sensibility.

The alcoholic group has yielded little information for present purposes, though perhaps the most important from a clinical point of view. It was found [30] that the chronic alcoholic invariably had a high threshold for this form of stimulation, and that this threshold fell after the withdrawal of the drug. In most of these cases (except those of Korsakow's psychosis and a few others) neuritis was not demonstrable by ordinary clinical methods or histologically in those that came to necropsy. It has thus been possible to demonstrate a distinct functional impairment in the receptor mechanism, by this method, without evidence of structural lesion. Apparently such a change in function can be induced by the administration of drugs to normal subjects, human or animal. E. L. Porter [31] has demonstrated increase in reflex irritability after the administration of strychnin to the experimental animal. Martin, Grace and McGuire [32] have shown a rise in this threshold in normal human subjects after the ingestion of moderate doses of acetphenetidin, and Dodge and Benedict [33] have shown a similar rise after varying doses of alcohol. The results obtained in the cases of Korsakow's syndrome showed that a severe, frank neuritis can exist without total impairment of conduction power, although these cases showed the highest values in this group, as was to be expected.

The psychoneuroses,[34] with the exception of the occupation neuroses and the traumatic neuroses, show a normal esthesia to faradism. In

29. Southard, E. E.: On the Somatic Sources of Somatic Delusions, J. Abnorm. Psychol. **7**:326, 1912-1913; ibid.: The Possible Correlation Between Delusions and Cortex Lesions in General Paresis, ibid. **8**:259, 1913-1914.

30. Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: IV, The Alcoholic Group, J. Nerv. & Ment. Dis. **45**:410, 1917.

31. Porter, E. L.: Variations in Irritability of the Reflex Arc. II. Variations Under Strychnin, Am. J. Physiol. **36**:171, 1915.

32. Martin, E. G.; Grace, C. M., and McGuire, J. H.: The Influence of Drugs on the Human Sensory Threshold, J. Pharmacol. & Exper. Therap. **6**:257, 1915.

33. Dodge, R., and Benedict, F. G.: Psychological Effects of Alcohol, Publication No. 232, Carnegie Institution of Washington, Washington, D. C., 1915.

34. Grabfield, G. P.: Variations in the Sensory Threshold for Faradic Stimulation in Psychopathic Subjects: V, The Group of Psychoneuroses, J. Abnorm. Psychol. **11**:328, 1916-1917.

such cases no structural pathology has ever been demonstrated. The occupation and traumatic neuroses are, sometimes at least, associated with demonstrable lesions. However, the results in the few cases of occupation neuroses that opportunity has permitted us to examine, seems to point to a possible division of these cases into those in which there is a true neuropathy or "functional neuropathy,"[35] and those of a purely psychogenetic nature. As to the traumatic neuroses, we have only seen two cases, both of which yielded definitely pathologic values by this test. We felt that these cases probably had an organic basis, even though they were quite negative to usual clinical methods of examination. The results in this group emphasize the possibility of obtaining, by this method, evidence of true functional impairment impossible of demonstration otherwise. Clinically, the results of this test are of importance in aiding in the differentiation of these cases from the true psychoses.

The epileptics, senile and syphilitic cases have not been considered, and will be taken up later together with the data derived from other cases showing definite neurologic lesions.

DATA

Data for the present study were obtained from 612 observations on 261 cases chosen at random from the admissions to the Psychopathic Hospital, Boston. Patients obviously unsuitable were not attempted.

In this group of cases (see Table 1), as in the first group studied, it is seen that the threshold of the psychopathic patient tends to be high, the value in the entire series averaging 197 beta units. This tendency of the psychopathic patient to have a faradic hypesthesia is not due to his mental incapacity as such. The only results here reported are those in which there was no doubt as to the validity of the reading, i. e., those in which conscious or unconscious deception on the part of the patient tested could be absolutely ruled out.

The Peripheral Neuron.—Cases such as those showing frank neuritides invariably yielded high threshold values. This is true whether there was in addition to the peripheral lesion a central lesion or not. As an example of the former are the cases presenting Korsakow's syndrome, and of the latter, cases of alcoholic neuritis without demonstrable central lesions.

As has been pointed out above, in many cases this method shows an hypesthesia in cases where such an impairment has not before been suspected. In the four cases of acroparesthesia studied (these are classified with the neurologic group in Table 1), we found uniformly

35. Southard, E. E., and Solomon, H. C.: Occupation Neuroses, in Handbook for Occupational Diseases, Ed. by Hanson, Washington, D. C.

high threshold values, confirming the suspicion from previous studies that the peripheral receptor system was impaired in this disease. Oppenheim [36] considers that this may possibly be a spinal cord condition, but that "it is probably due to a condition of irritation in the vasomotor centers, by which the arteries are contracted and the nutrition of the sensory nerve endings in the extremities are impaired. The same author also describes these cases as being negative to sensory tests or showing "merely slight decrease of the sensibility of the finger tips."

TABLE 1.—Data Available in 261 Cases Studied *

Diagnosis	Number Studied	Number of Observations	Limits of Variation in Beta Units	Average
Delirium tremens	22	32	85- 493	241
Acute alcoholic hallucinosis	16	25	46- 545	269
Korsakow's psychosis	6	18	156-1500	443
Unclassified alcoholic psychoses	14	20	86- 600	249
Dementia praecox	51	87	61- 777	186
Manic depressive insanity	25	66	23-1061	237
Psychoneuroses	30	113	41- 785	179
Epilepsy	8	12	108- 408	217
Senile group	7	11	124- 738	274
Defective group	12	22	65- 364	179
General paresis	18	50	41- 589	271
Huntington's chorea	2	4	141- 650	307
Neurologic	20	104	38-1881	292
Disturbances of the internal secretions	4	6	115- 234	147
Unclassified	26	42	66- 621	210
Total	261	612	23-1881	197

* The averages in the table are without any corrections for wrong diagnoses or complicating conditions, such as alcoholism, which would tend to raise or lower the threshold, thus throwing such a case out of alignment with its group.

From the study of this group we may safely conclude that lesions of the peripheral neuron cause a faradic hypesthesia, though we have not yet encountered clinically a faradic anesthesia from such a lesion, no matter how severe.

The Spinal Neuron. — Cases showing lesions of the posterior columns have for the most part high threshold values. We have examined five cases of this type: two of tabes dorsalis, two of taboparesis and one of ataxic paraplegia. Case No. 4656 [37] showed clinically an hypesthesia over the ulnar distribution of both hands and a hyperalgesia over both arms. The grip of the right hand was stronger than that of the left. Using the little and ring fingers the threshold value on the left was 326 and on the right 574 beta units; using the index and middle fingers (these were the ones used throughout this work unless otherwise stated) the threshold on the left was 159, and

36. Oppenheim, H.: A Textbook of Nervous Diseases, Ed. 5, 1911. Trans. by Alexander Bruce, New York, 1911.

37. Case numbers refer to the record numbers in the Psychopathic Hospital, Boston.

on the right 383 beta units. Thus it is seen that in this case the diminution in sensory acuity on the ulnar side is accompanied by a diminution in faradic sensibility. The values are higher on the side of the body showing the greater muscular power in this case. Case No. 3931, diagnosed as taboparesis, showed diminution of sensibility to pain over the legs and chest, but no sensory changes over the arms. This case showed a threshold of 433 beta units. However, the cortical involvement in this case must be borne in mind. The case of ataxic paraplegia studied showed a threshold of 178 on the right and 143 on the left. Cutaneous sensation was clinically unimpaired. The reflexes were greater on the left than on the right, but it was noted that this is a doubtful finding. The case seems to make negative the effect of posterior column sclerosis, per se, in raising this threshold. The lesions in the spinal ganglion in tabes may well be the chief cause of the raised threshold in these cases, assuming that such lesions most probably affect more than the fibers of deep sensibility.

TABLE 2.—FIGURES IN BETA UNITS OBTAINED FROM EXAMINATION OF ALL THE FINGERS IN PAIRS OF A CASE SHOWING RESIDUA FROM AN ATTACK OF BOTULISM

Fingers Used	Right	Left
Thumb and index	282	127
Index and middle	293	128
Middle and ring	252	163
Ring and little	255	217
Little and thumb	361	214
Average	289	170

Further evidence as to the spinal path is obtained from the study of a case showing certain residua following an attack of botulism. The case has been reported [38] elsewhere. At present this patient has a hemithermo-anesthesia on the right including the entire body up to the ramus of the jaw, above which sensation is normal. On this side there is also a slight hyperalgesia and a slight hyperhidrosis. Extremes of heat and cold are felt as pain. On the left the threshold of all the fingers taken in pairs (see Table 2) averaged 170 beta units and showed an increasing hypesthesia as the ulnar side was approached. The right hand, on the contrary, similarly tested averaged 289 beta units, the lowest value obtained on this hand being 35 beta units above the highest on the left. This patient was not only not psychopathic but was trained in introspective observation. Subjectively he made the interesting

38. Stiles, P. G.: A Case of Apparent Food-Poisoning of the Type Known as Botulism or Allantiasis, J. A. M. A. **41**:2301 (Dec. 27) 1913.

observation that on the left the sensation was localized in the ends of the fingers, whereas on the right it was located between the two fingers tested and felt more like a muscle twitch.

Cases of Syringomyelia.—The results of two cases of syringomyelia tested in various ways properly fall in this section.

Case 4166.—P. W., aged 40, had no mental symptoms except irritability of temperament. Blood pressure was: systolic 115, diastolic 75. The Wassermann tests of blood and spinal fluid were negative. The pupils showed a slight horizontal nystagmus, especially the left. They reacted normally to light and accommodation. Biceps and triceps reflexes were absent. The abdominal reflex was absent. The Babinski and Oppenheim signs and ankle clonus were present on both sides. There was patellar clonus on the right. Knee jerks and ankle jerks were equal and exaggerated on both sides. The cremasteric reflex was absent. There was slight swaying in Romberg's position. Tactile sensation (light touch) was everywhere unimpaired. Temperature and pain sensation were lost everywhere except over the face. Ordinary clinical laboratory tests were essentially negative.

Case 3729.—K. M., aged 54. The only mental symptoms that this patient showed were some persecutory delusions. Blood pressure was: systolic 170, diastolic 110. The Wassermann tests of the blood and spinal fluid were negative. There was marked tremor of the extended hands; marked scoliosis with convexity to the right—a true rotolateral curvature. Pupillary reactions were normal. The conjunctival reflex was absent. Biceps and triceps reflexes were absent. The abdominal reflex was absent. The knee jerks and ankle jerks were hyperactive but equal. Babinski's sign was absent. Oppenheim's sign was inconstant. There was ankle clonus on both sides, better sustained on the right. Romberg's sign was absent. Tactile sensation (light touch with cotton) was everywhere unimpaired. On the right side, however, she was unable to distinguish the head from the point of a pin. There was temperature anesthesia over the right arm and hand, and an apparent slight disturbance everywhere on both sides. Pain sense was absent on the right side and somewhat disturbed on the left. Deep sensation was well preserved everywhere. Muscle sense was poor. There was slight dysdiadokokinesia on the right. Ordinary clinical laboratory tests were essentially negative.

A number of observations (see Table 3) on both these cases show that Case 4166 had an average threshold on the right of 396 beta units as compared with 505 beta units on the left. Case 3729 averaged 928 beta units on the right and 472 on the left; the latter showed comparable figures in one observation made on the feet. It is interesting to note that both of these patients could distinguish painful faradic stimuli from threshold stimulation. These were roughly estimated on Case 3729 in the feet and were found to be 875 beta units on the left and 4,000 on the right. As a comparison, it is to be noted that the strength of stimulus at the threshold for these cases is, when applied to a normal person, distinctly painful.

Cases of Cortical Lesions.—In this section we will consider those patients whose lesions are practically wholly cortical with evidence of a definite preponderance of damage on the one side or the other. In the cases of general paresis (see Table 4) there was no sensory impairment demonstrable by the usual clinical methods. It will be seen from the table that the more affected side averages 333 beta units as compared with 175 on the less affected side. In every case the threshold of the more affected side is higher than on the less affected side. Case P. B. has the following history:

TABLE 3.—Observations on Two Cases of Syringomyelia. No. 3729 Had Clinical Evidence of Advancement of the Process in the Period of Observation

Case Number	Parts Tested	Right	Left	Date
3729	Index and middle fingers	845	517	10/28/14
3729	Index and middle fingers	513	131	11/ 2/14
3729	Index and middle fingers	706	265	11/ 9/14
3729	Index and middle fingers	794	333	11/17/14
3729	Index and middle fingers	345	195	11/30/14
3279	Index and middle fingers	...	246	12/ 6/14
3729	Index and middle fingers	1590	585	12/10/14
3279	Index and middle fingers	692	679	1/ 2/15
3729	Index and middle fingers	1881	1302	1/ 3/15
3729	Feet	989	585	12/10/14
	Average	928	472	
4166	Index and middle fingers	302	500	1/11/15
4166	Index and middle fingers	462	829	1/12/15
4166	Index and middle fingers	418	307	1/14/15
4166	Index and middle fingers	379	335	1/16/15
4166	Index and middle fingers	418	444	1/17/15
	Average	396	505	

TABLE 4.—Cases of Cortical Lesions

Case Number	Diagnosis	Threshold in Betas Right	Threshold in Betas Left	Side More Affected
3920	Imbecile	119 105	172 120	Left
3999	Cerebrospinal syphilis	270	189	Right
3822	General paresis	271	196	Right
3832	General paresis	182	255	Left
3674	General paresis	370	200	Right
3857	General paresis	275	215	Right
P. B.	Cerebral thrombosis	400 843	150 158	Right

He was a man of 50, who in the early part of 1912 developed a spastic paralysis of the right side. A tumor in the left motor area was suspected. He was operated on and at operation a large mass of obviously extraneous tissue was removed from the left cortex. The process, it was seen, had also involved the postcentral gyrus. On pathologic examination it was found that this mass was composed of scar tissue, and it was decided that the process was in all probability an extensive cerebral thrombosis, apparently quite sharply localized to the left side. He made an excellent operative recovery, and went home in July, 1912. In December, 1914, he noticed a slight increase in the symptoms on the right. The left side gave him no trouble throughout the period that he was under observation.

This patient, when first tested in 1912, showed a threshold of 150 beta units in the left hand and 400 in the right. In 1914, on his return, he showed a threshold of 158 beta units on the left and 843 on the right. In addition to the above, a number of patients have been tested who showed diffuse cortical lesions. These showed invariably a high threshold value and include the senile group, huntington's chorea, various syphilitic lesions including other cases of general paresis than those mentioned above (see Table 1). These cases will be discussed later.

DISCUSSION

It was to be expected that it could be shown that faradic stimuli used one of the paths described by Head, Rivers and Sherren.[39] An absolutely dogmatic statement as to the path of these impulses is not warranted by the evidence now at hand, and further work should be done to firmly establish such a path.

Several hypotheses suggest themselves. Faradic stimuli may use all paths equally and therefore the impairment of any sensory path may raise the threshold value to this form of stimulation. However, there are serious objections to this in that the hypesthesia caused by lesions of pain and temperature fibers far exceeds that in which only other systems are involved. This suggests the hypothesis that such fibers are the path normally used by these stimuli. However, the finding of some sensory impairment in many cases considered chiefly motor would suggest the possibility of the path associated with the motor nerves, i. e., the fibers conveying deep sensibility. Such an hypothesis is supported by the findings in cases of peripheral neuritis showing almost wholly motor symptoms, such as contractures, paralyses, etc. Another implication is found in the cases showing posterior column sclerosis which invariably showed an hypesthesia.

Our feeling is that the degree of hypesthesia associated with lesions affecting the thermal fibers, as shown in the cases with dissociation of sensation, is so marked as compared with that caused by apparently equal destruction of other sets of fibers that we are justified in concluding that faradic sensibility is carried largely, if not wholly, by these fibers in the lateral columns.[40] The hypesthesia found associated

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39. Head, H.; Rivers, W. H. R., and Sherren, J.: The Afferent Nervous System from a New Aspect, Brain, **28**:99, 1905. Head, H., and Sherren, J.: The Consequences of Injury to the Peripheral Nerves in Man, Ibid., p. 16. Head, H., and Holmes, G.: Sensory Disturbances from Cerebral Lesions, Ibid., **34**:102, 1911-1912. Rivers, W. H. R. ,and Head, H.: A Human Experiment in Nerve Division, Ibid. **31**:323, 1908.

40. Head, H., and Thompson, T.: The Grouping of Afferent Impulses in the Spinal Cord, Brain **29**:537, 1906. May, W. Page: The Afferent Path, Ibid., p. 742.

with other spinal lesions can be explained by the statement that these fibers may be affected without clinical evidence and that the delicacy of this reaction has discovered hitherto unsuspected involvement. This is not too great an assumption in view of the fact that impairment of sensation has been demonstrated by this method in patients thought to have normal sensibility. We cannot exclude the other fibers as a path in stimuli of great intensity, as there may be a spread or a change in reaction — each of which possibilities has analogues in the behavior of the nervous system. We have not mentioned the possibility of a separate set of fibers carrying this sensation as the teleological argument against such fibers is practically conclusive. We believe, therefore, that the evidence points strongly to the thermal fibers as the path of this form of stimulation.

Presumably, then, we should find evidence that the postcentral convolution is the arrival platform for the impulses that we are studying. The evidence on this point, while mostly indirect, is, we believe, conclusive. That this gyrus contains the sensory projection areas was shown experimentally by Cushing[41] (see also Bergmark[42]). We have derived evidence on this point from a study of a large variety of cases showing various cortical pathology, some diffuse and some localized. Those cases showing widespread general cortical pathology invariably showed high threshold values.

The cases of epilepsy studied (see Table 1) showed an average threshold of 217 beta units — a marked disturbance of the receptor mechanism for this form of stimulation which cannot be accounted for by peripheral lesions. Unfortunately, the evidence of cortical pathology is not conclusive. Southard has studied the glia tissue in epilepsy,[43] and he suggests that the epileptic discharge really depends on the simplification of the course of the sensorimotor afferent impulses by a reversion of this synapse to the spinal type. This may be correlated with our results in so far as they indicate the possibility of increased synaptic resistance between afferent impulses and the paths leading to the nonmotor areas of the cortex, and thus imply the possibility of lowered synaptic resistance to impulses from the periphery to the motor cortex. The few normal values obtained in this group may be correlated with the possible true idiopathic epilepsy.[44] However, while most

41. Cushing, H.: A Note on the Faradic Stimulation of the Post cerebral Gyrus in Conscious Patients, Brain **32**:44, 1909.

42. Bergmark, G.: Cerebral Monoplegia with Special Reference to Sensation and to Spastic Phenomena, Brain **32**:342, 1909.

43. Southard, E. E.: On the Mechanism of Gliosis in Acquired Epilepsy, Am. J. Insan. **64**:607, 1908.

44. Thom, D. A., and Southard, E. E.: An Anatomical Search for Idiopathic Epilepsy, Rev. Neurol. & Psychiat. **13**:471, 1915.

of the research up to the present has been unsatisfactory,[45] the cause of this condition is likely to be found in the abnormal excitability of the efferent path. If this proves to be true, an efficient reaction of the organism would be a decreased excitability of the afferent path or an increase in the resistance of the synapses between the two in the cortex. It seems not unlikely that the faradic hypesthesia here demonstrated represents the effects of some such protective reaction.

In the senile group of psychoses high values have been found. These cases are usually associated with gross brain lesions at necropsy. In previous work, however, we have gained the impression that there is a decreasing sensitivity with increasing age (though not to what we have considered pathologic values). Such an hypesthesia as this may be accounted for by impaired nutrition of nervous tissue either in the periphery or centrally either due to frank arteriosclerosis or to the "narrowing of the capillary field" which Mackenzie [46] has pointed out.

Another small group of cases in which there is generalized cerebral involvement is that of Huntington's chorea. Oppenheim [36] states that there are "disseminated, miliary, encephalitic foci especially in the cortex and the motor region," and Spiller [47] noted a proliferation of the neuroglia, especially in the second and third layers of the cortex chiefly in the motor area. He also conveys the impression that this is a diffuse process. These cases showed a constantly high threshold, and with such cerebral findings at necropsy the possible relation of the effect of lesions of what are probably associational regions on sensation, seems worth further study.

In respect to general cerebral conditions, we have had the opportunity of examining two cases in which the clinical signs were only of increased intracranial pressure. Both these cases showed a high threshold value on both sides without preponderance of hypesthesia on the one side or other. The one proved to have a left cerebellar tumor and the other a large glioma occupying chiefly the midbrain.

From the foregoing paragraphs it is seen that general lesions of the cortex and various changes of the relations inside the cranium apparently cause an hypesthesia. The cases mentioned which showed a preponderance of lesion on one side or the other and a corresponding preponderance of hypesthesia complete the evidence that the postcentral cortex is the arrival platform for these impulses. In addition, there is

45. Vogt, H.: Epilepsie, in Handbuch der Psychiatrie, Spezieller Teil 1ste Abteilung, Ed. by G. Aschaffenberg, 1915.

46. Mackenzie, James: Diseases of the Heart, Ed. 3, London, 1914.

47. Spiller, W. G.: Symptomatology, Pathology and Treatment of Choreiform Movements, J. A. M. A. **44**:433, 1905.

evidence concerning this in the study of the dementia praecox group which brings out a striking parallelism between the postrolandic lesions discovered in catatonic cases by systematic brain photography [48] and high threshold values.

We have, then, evidence that threshold faradic sensation is carried by the peripheral neuron following the path of the thermal fibers in the cord and arriving at the cortex in the postcentral gyrus. Lesions of any portion of this path produce an hypesthesia to faradic stimulation.

In conclusion, we wish to emphasize the possibilities of further study of the nervous system by this method, both experimentally and clinically, as a means of throwing some light on the variations in the reactions of the nervous system under various physiologic and pathologic conditions, the latter whether structural or functional.

SUMMARY AND CONCLUSIONS

1. The method of Martin for determining the strength of shocks delivered by the secondary coil of an inductorium in absolute units is described as used by us in the determination of the sensory threshold for faradic stimulation in 261 cases (612 observations). The advantages of this method over previous methods is pointed out.

2. Previous work with this method is reviewed.

3. Clinical considerations have been purposely omitted from this discussion, but it is to be noted that the epileptic, senile and syphilitic groups of cases tend to have a faradic hypesthesia. These groups have not been reported elsewhere.

4. Cases of acroparesthesia and Huntington's chorea, though few in number, showed constantly a pathologically elevated sensory threshold value in beta units, i. e., the number of units of shock required to cause sensation.

5. Certain suggestions as to the mechanism of the relation of the variation of sensibility with that of emotional tone in the cases of manic-depressive insanity have been made.

6. From a study of this pathologic material, the hypothesis is suggested that faradic stimuli use the path of the thermal fibers in the cord and presumably in the peripheral nerves, arriving in the cortex at the postcentral gyrus. Lesions affecting any part of this path either

48. Southard, E. E.: On the Direction of Research as to Analysis of Cortical Stigmata and Focal Lesions in Certain Psychoses, Trans. Assn. Am. Physicians **29**: 1914.

functionally or structurally cause an hypesthesia to this form of stimulation. It was impossible to determine whether such stimulation used the thermal receptors in the skin.

It is a pleasure to acknowledge my indebtedness to Dr. E. G. Martin for his help and to Drs. H. A. Christian, H. Cushing, W. E. Paul, and E. W. Taylor for the privilege of examining certain cases reported here.

In conclusion I wish to express my sincerest thanks to the late Dr. E. E. Southard, not only for placing the clinical material at my disposal, but also for his kindly interest and invaluable and stimulating suggestions throughout this work.

University Hospital.

A SIMPLE METHOD FOR RECORDING CHANGES IN CUTANEOUS SENSIBILITY*

LAURENT FEINIER, M.D.
NEW YORK

The New York Neurological Institute and several other New York hospitals have adopted a method for the recording of changes in cutaneous sensibility, which on account of its simplicity is, we believe, worthy of being used as a standard. The method was suggested by Dr. Elsberg, and has served in our institution to obviate the difficulties often encountered in the understanding of the records of sensory disturbances. Whenever such a chart is examined, whether it be at the bedside or in a medical journal, it often requires considerable study before the meaning of the markings by which the different disturbances of sensibility are characterized are understood.

The sensory sheets we use at the New York Neurological Institute are of a uniform size to fit the hospital bedside chart, i. e., 8 5/16 inches wide by 10 11/16 inches long. The chart has printed on it a human figure in the anterior and posterior position (see chart). The charts are printed on paper of three colors—the white for the tactile sensation, the red for temperature and the yellow for pain. In the upper right hand corner of the sensory sheet of appropriate color is printed the sensation to be recorded. Below the body outlines is printed a key to the method of making sensory markings so that a physician who is not accustomed to making the records according to the standard can easily understand how to record the sensory disturbances.

For total loss of sensation transverse lines are made and, as transverse lines mean lost sensation, it is of litttle consequence how near together or how far apart the lines are drawn. It has, however, been found advantageous to make the lines 1/8 to 1/4 inch apart to facilitate reading. Thus, transverse lines drawn on the figures of the white sensation sheet mean anesthesia; transverse lines drawn on the figures of the red sheet (temperature) mean thermal anesthesia; transverse lines drawn on the figures of the yellow sheet (pain) mean analgesia.

Diminished sensation is represented by three varieties of oblique lines running from the right downward and to the left; for slight diminution these oblique lines are spaced 1/4 inch apart; for moderate diminution 1/8 inch apart, and for marked diminution 1/16 inch apart. It is not to be hoped that mathematical preciseness in spacing the lines exactly according to the above measurements will always be maintained,

* From the Clinics of the Neurological Institute, New York.

but if they are approximated a quick survey should convey an intelligent grasp of the sensory situation. Thus, oblique lines mean diminished sensation, and the nearer together the lines are the more marked is the diminution in sensation. As it is often advisable to record separately disturbances in the recognition of heat and cold, oblique lines running from right to left and downward are used to denote disturbances in the recognition of heat, and those running from left to right and downward are used to denote disturbances in the recognition of cold.

In a similar way, vertical lines show hypersensitiveness to touch, pain or temperature. The more closely together the vertical lines are, the more marked is the hypersensitiveness; thus, the vertical lines on the white sensation chart record the presence and degree of hyperesthesia; vertical lines on the yellow chart record the various degrees of hyperalgesia and those on the red chart hypersensitiveness to hot and cold.

The accompanying three charts, on which are recorded the sensory disturbances of the integument exhibited by a patient with a spinal cord tumor in the upper dorsal region, will serve as an illustration of the method used to record sensory changes.

On the white or tactile chart, it will be noted, is an anesthesia of the lower extremities extending almost to the umbilicus anteriorly and to the level of the first lumbar vertebra posteriorly. Anteriorly, above the lost sensation, there is over the upper abdomen and lower thorax an area of slight diminution in tactile sensation, and just below the left nipple an area of marked diminution. Posteriorly, sensation is lost completely to the seventh dorsal vertebra on the right with an area of moderate diminution from the level of the sixth dorsal vertebra to the first lumbar on the left.

On the yellow or pain sheet, there is an analgesia of the lower extremities with an area or band of slight hyperalgesia posteriorly at the level of the angles of the scapulae, and between this and the analgesia on the right posteriorly an area of slight hypalgesia.

Anteriorly similar changes are noted, and there is in addition a band of moderate and slight diminution in pain sense extending from the left to the right, and above this a band of moderate hyperalgesia.

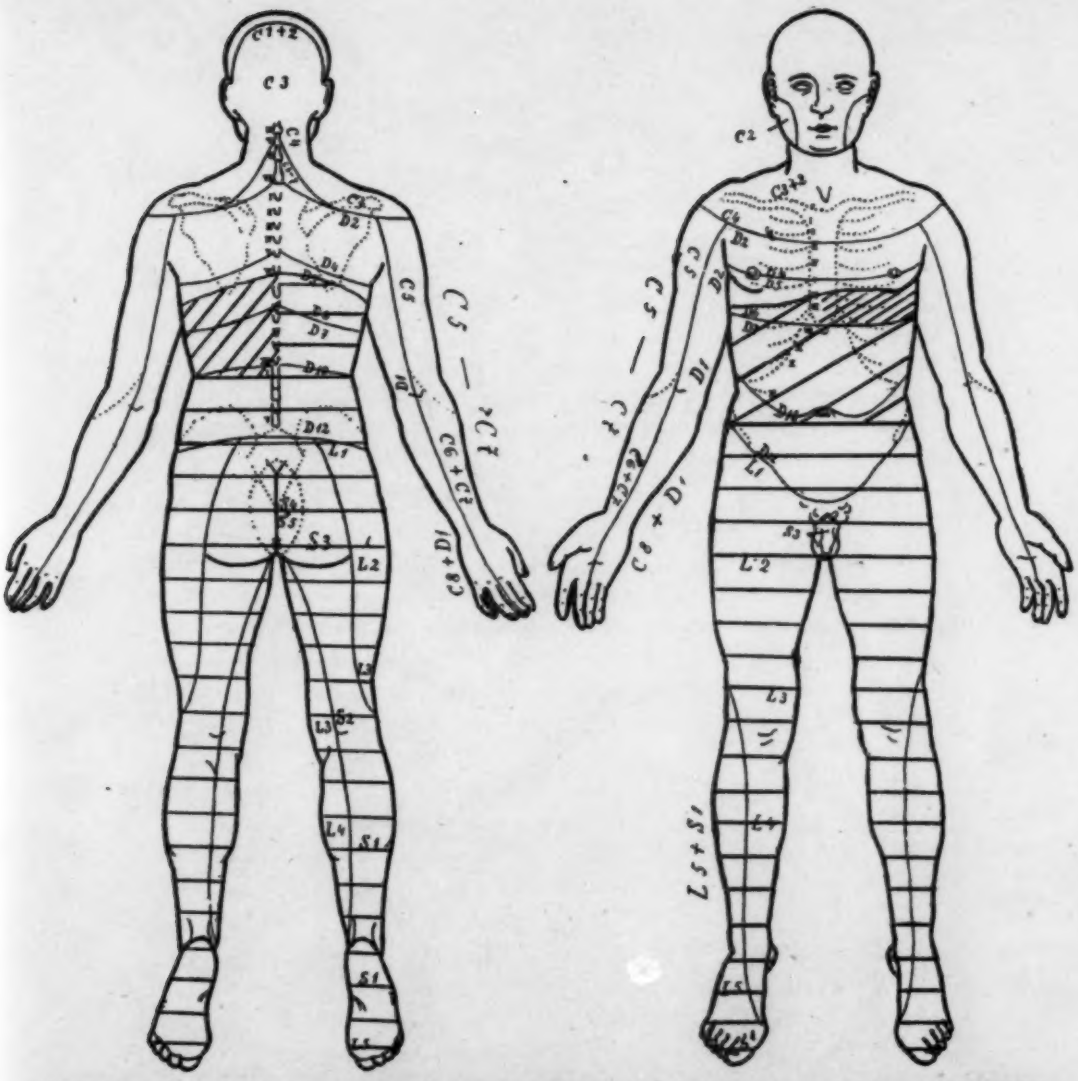
Similarly, on the red or thermal sense chart there is recorded a total loss of sensation in the lower extremities, above this an area of moderate diminution, and extending around the figure above the areas of lost and diminished thermal sense, there is a band of moderately hypersensitive thermal sense.

149 East Sixty-Seventh Street.

TACTILE

THE NEUROLOGICAL INSTITUTE, New York

Name: Mr. A. Date: Oct. 18, 1919.



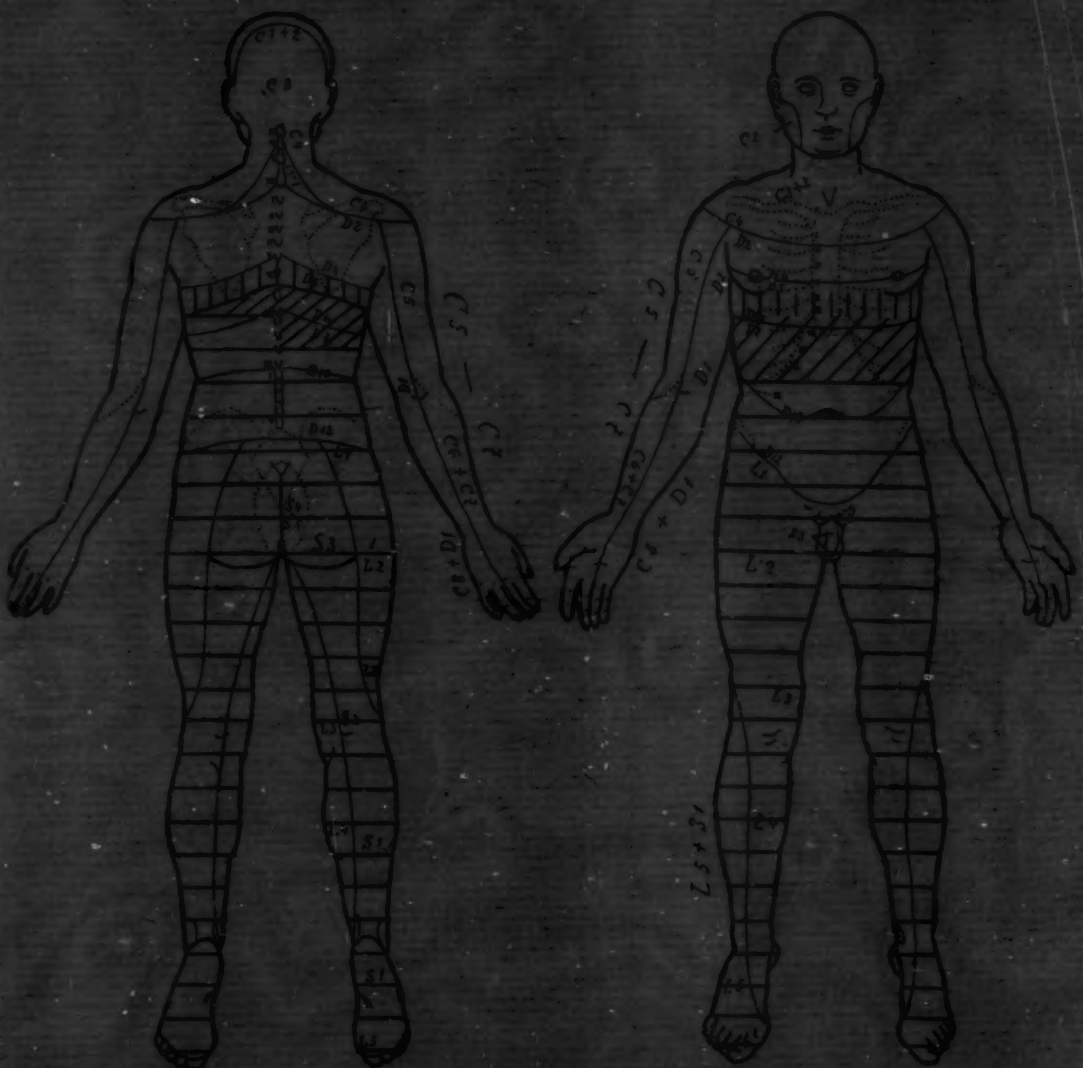
Sensation lost | Slight diminution | Moderate diminution | Marked diminution | Slight increase | Moderate increase | Marked increase

TEMPERATURE

THE NEUROLOGICAL INSTITUTE New York

Name: Mr. A.

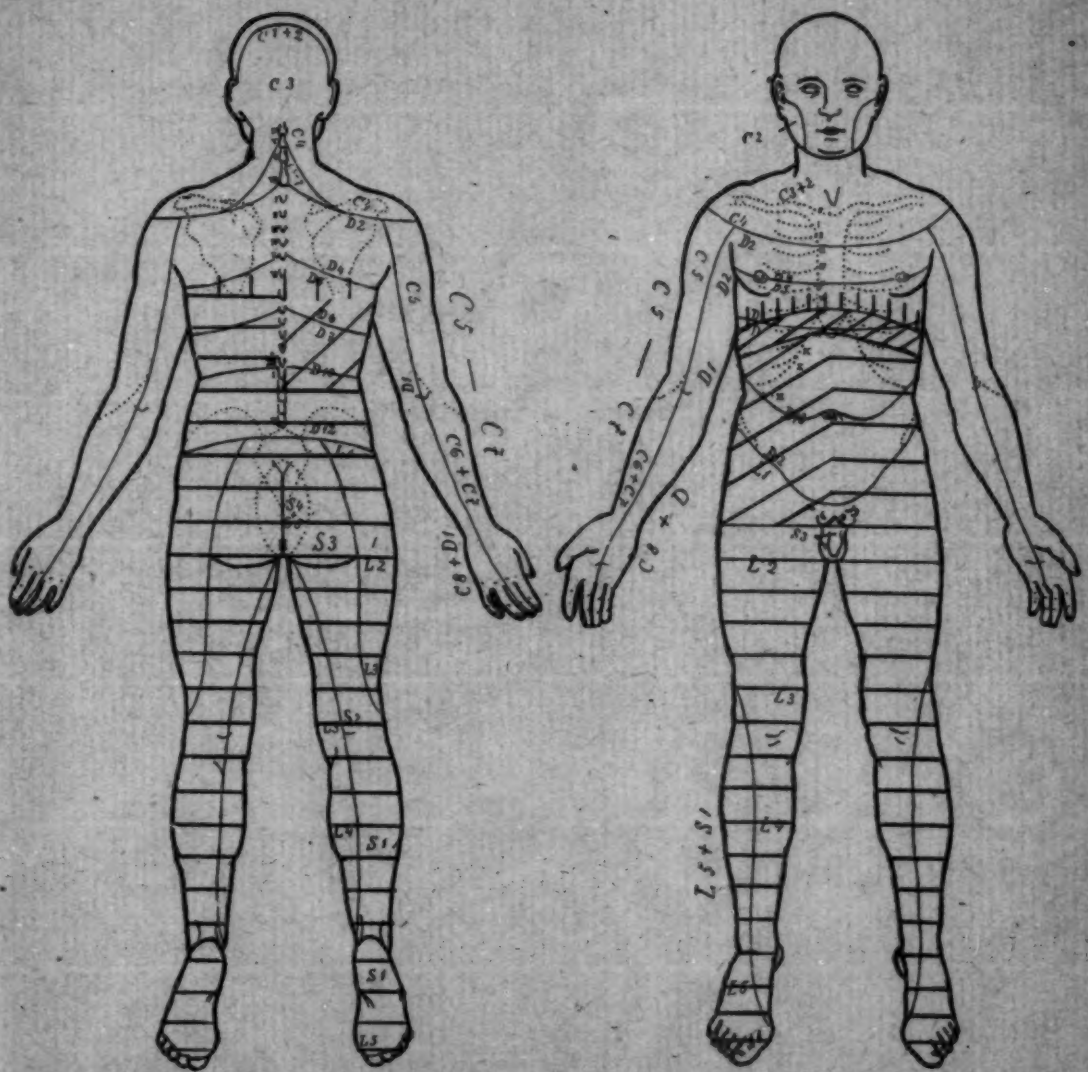
Date: Oct. 18, 1919.



PAIN

THE NEUROLOGICAL INSTITUTE, New York

Name: Mr. A. Date: Oct. 18, 1919.



Sensation lost | Slight diminution | Moderate diminution | Marked diminution | Slight increase | Moderate increase | Marked increase

News and Comment

AMERICAN NEUROLOGICAL ASSOCIATION ELECTS OFFICERS

At the last meeting of the American Neurological Association, June 1, 2 and 3, the following officers were elected: president, Dr. Sidney I. Schwab, St. Louis; first vice president, Dr. Hermon C. Gordinier, Troy, N. Y.; second vice president, Dr. Henry H. Donaldson, Philadelphia; secretary-treasurer, Dr. Frederick Tilney, New York; councilors, Drs. J. Ramsay Hunt, New York and Henry M. Thomas, Baltimore; councilors holding over, Drs. E. W. Taylor, Boston, D. J. McCarthy, Philadelphia, and T. H. Weisenburg, Philadelphia. The time and place of the next meeting will be determined by the council about next February.

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

A new association under the above title has been organized through the initiative of the New York Neurological Society.

Its membership was originally to have consisted of all members in good standing of the neurological societies of the cities in fairly close proximity to New York, namely, Baltimore, Boston, Brooklyn, Philadelphia and Washington. However, invitations have since been extended to the neurological societies of the cities beyond these limits, and preliminary favorable responses have been received from Chicago, Pittsburgh and San Francisco.

The Organization Committee is composed of the following: Drs. Pearce Bailey, Charles L. Dana, J. Ramsay Hunt, Smith Ely Jelliffe, George H. Kirby, Foster Kennedy, Bernard Sachs, Israel Strauss, Frederick Tilney, Walter Timme, Lewellys F. Barker, E. W. Taylor and T. H. Weisenburg.

The aim of the organization is to foster research in neuropsychiatric diseases. Its meetings will be held yearly and are to be conducted by a commission whose members will act as an investigating body for the presentations and theses submitted at each annual meeting. The subject for each annual meeting will be announced from one to three years in advance in order to enable the necessary laboratory and other research work to be conducted.

At each annual meeting there will be but one subject for discussion, all phases of which will be covered and all discussion and all activity at the meeting will be directed toward the solution of the problems presented. At the conclusion of the annual meeting the commission will prepare and publish such data and views presented at the meeting as they deem fit, with their own interpretation of the various papers presented. Such publication is to be of broad and comprehensive scope.

The Commission has decided on New York for the first meeting, during the Christmas week of 1920. The subject for discussion is "The Acute Nonsuppurative Infections of the Nervous System." Already a more or less complete program has been decided on by the Program Committee, of which Dr. Charles L. Dana is Chairman.

The financial burdens will be borne by the members. Each member is to pay a fee of $5 yearly to the Secretary-Treasurer of his local neurological society, who will transmit all these fees by October of each year to the Secretary-Treasurer of the Research Association, Dr. Foster Kennedy. This fee will entitle the member to be present at the annual meetings and probably also to receive a copy of the transactions when these are completed.

The organization of the Research Association is practically completed and the details will be presented at the business session of the annual meeting next December. Dr. Walter Timme is chairman of the Organization Committee and Dr. Frederick Tilney is chairman of the Committee of Arrangements for the first annual meeting.

Abstracts from Current Literature

ZUR PATHOLOGIE DER EPILEPSIE NACH SCHADELSCHUSS-VERLETZUNGEN (PATHOLOGY OF EPILEPSY FOLLOWING GUNSHOT WOUNDS OF SKULL). E. Redlich, Ztschr. f. d. ges. Neurol. u. Psychiat. 1:48, 1919.

Redlich reports fifty-seven cases of epilepsy following head injuries of warfare. In eighteen of these the attacks were designated as early attacks, occurring at the time of injury or soon following operation; of the remaining thirty-nine cases in which late attacks occurred, twenty-six developed in the first six months (66.7 per cent.), and eight cases developed in the second six months (20.6 per cent.), making a total of 87.3 per cent. within the first year. The frequency of the onset of the late attacks during the first six months has been emphasized by Voss and Jolly. A survey of the literature of traumatic epilepsies of warfare is made with especial reference to a statistical study of frequency of occurrence.

Epilepsy occurring after war injuries is characterized by relatively infrequent attacks; the author has not observed a status epilepticus. Among his series he reports one case of a single attack occurring early after trauma; in three others, single late attacks, occurring fifteen, eighteen and twenty-six months later, respectively.

The disposition for epilepsy is the head injury. More frequent than in ordinary epilepsy, the exciting factors are evident. Most important is the direct irritation of the motor cortex, producing typical jacksonian epilepsy. These attacks frequently cease after surgical removal of the irritating factors, bone fragments, bullet particles, hemorrhage, etc., but they may continue or recommence after an interval of time. Attacks may occur during or following surgical intervention, not directly involving the motor cortex (Böttiger). Redlich reports a considerable number of cases in which the attacks followed early operation. He interprets these attacks as due to mechanical irritation of the motor zones, due to the trauma of operation, hemorrhage in the subarachnoid space and in the cortex, etc.

Although early operation, débridement, removal of fragments, have been generally recommended to prevent future attacks, in the experience of the writer this procedure cannot be considered as offering certain protection, because splinters, and especially metal splinters, cannot be entirely removed by surgical procedure, even by a secondary operation. The covering in of a bone defect following skull injury is frequently followed by epileptic attacks. This procedure has been suggested as preventing such attacks. Other exciting causes are: inflammatory reaction of the wound, sensory stimuli to parts involved in the epileptic contractions, such as application of the faradic current in recent hemiplegias, massage, passive motion and undue amount of exercise; local application of heat over the traumatic area and exposure to the sun's rays. Absolute abstinence from alcohol should be the rule in these cases.

Most often the epileptic attacks are of the jacksonian type, although all forms are observed, such as the form beginning with jacksonian epilepsy and becoming generalized with loss of consciousness. Attacks of vertigo, with

sensations of heat over the body, nausea and transient loss of consciousness, is a symptomatology frequently met with. Psychic equivalents were not observed, and postparoxysmal psychic disturbances were comparatively rare. Aurae of motor and sensory origin were frequent, and would often give timely and useful warning for protection or even abortion of the attack. In the jacksonian type it appears that there is a predisposition for the onset of the spasm in certain anatomic regions; in the author's cases the onset was much more frequent in the upper extremity. In the cases of organic hemiplegia the onset was in the extremity or parts of an extremity relatively less affected by paralysis; a completely paralyzed part has been observed to be free from spasm. The jacksonian spasm is typically a clonic spasm, although circumscribed muscle spasm of tonic form has been observed. When the jacksonian spasm extends it progresses in such a manner that the muscles are progressively affected in the order of their cortical representation. Consciousness is retained or is only slightly clouded. Following the epileptic attacks there may be transitory aphasia (exhaustion palsy); also temporary motor paresis of a member (paralytic equivalent). Extra paroxysmal twitchings in certain muscle groups, which recall either voluntary movements, or choreiform and athetoid movements are occasionally observed. Epilepsia continua or long continued rhythmic muscular contractions, from six to eight in a second, was observed in one case.

In fifty of the cases a brain lesion was evidenced by sensory-motor symptomatology, and in forty-eight cases there was a more or less pronounced affection of the motor region. From the psychic side there were episodic changes of disposition lasting one or more days: irritability and sudden outbreaks of temper were characteristic. Posepileptic psychoses have not been observed in the series. Only occasionally characteristics of chronic epileptic character were observed—circumstantiality, littleness, pedantry, narrowness of the thought content and general interest, egocentric psyche, exaggerated politeness and religious tendency. Regarding the intelligence, memory attention and apperception were particularly affected; arithmetic was strikingly difficult, and fatigability or mental effort was marked. In the association tests delayed reaction time, missed reaction, tendency to perseveration (repetition of the test word), and sentence formation instead of word pairing were frequent phenomena. However, no striking differences were noticed in these traumatic epilepsies from the idiopathic forms, or severe head traumas uncomplicated with epilepsy.

Research directed toward the cardiovascular system corroborated the earlier observation of Goldstein that bradycardia was frequent, especially in the reclining posture; in one case the rate was found to be 36. Bradycardia was found, however, in cases of brain injury without epilepsy. The blood pressure as a rule was low—in the majority of cases below 130 (Riva-Rocci). The blood picture revealed a leukopenia before the attack and a leukocytosis following, lasting a number of hours and reaching a count of 16,500 in one case (five hours after the attack). This same change in the blood picture has been found in poisoning by Witte peptone and is characteristic of anaphylactic shock. This would suggest that the epileptic attack is accompanied by an intoxication of protein nature.

Regarding the question of disposition to attack, the author from his experience in these traumatic cases holds firmly to his former thesis that any brain will react by an epileptic attack, provided the damage or noxus be sufficiently intense. In twenty-nine cases in which a thorough investigation

of the personal and familial hereditary history of the case was obtainable, only eight cases suggested possibility of an underlying predisposition in the affected person. Reflex epilepsy is excluded in the series; rather is the cause to be found in the brain lesion itself. The real problem in the traumatic epilepsy is to explain the cases in which the late attacks occur, namely, those cases in which an appreciable interval elapses between the trauma and onset of attacks. A small number may be explained by abscess formation or other local processes, such as plaques jaunes the result of trauma and contusion by blunt instruments. Nonpenetrating wounds by fracture may produce diffuse lesions of the brain causing general epileptic attacks.

The influence of the localization of the skull injury is discussed in detail with reference to available statistics which show that injuries of the parietal region, whether or not complicated by epilepsy, make up from 40 to 45 per cent. of all cases of skull injuries of warfare. In the author's series of epilepsy cases, however, there were thirty-seven cases, or a percentage of 63.7, with parietal wounds. In forty-two cases without epilepsy there were nineteen, or 45.3 per cent., with parietal wounds. The proportion of parietal cases with and without epilepsy would be 63.7 : 45.3. It is concluded that lesions of the parietal region of the skull have a particular signification for epileptic attacks, especially jacksonian attacks. As to the character of the lesion, it is particularly gutter, longitudinal and segmental wounds which are followed by epilepsy, as these wounds heal with large pulsating bone defects. In regard to the covering of defects, opinions vary greatly on this subject. Epilepsy is more frequently found in cases with than without bone defect, which is in part explained by the severe accompanying pathologic process affecting the brain substance itself.

When intracranial pressure is studied, bone defects are of especial interest. It is recalled that Kocher believed that epileptic attacks were caused by variation of intracranial fluid pressure and resulting disturbances of circulation in abnormally irritable cortical areas. A decompression operation with excision of the dura, forming a permanent valve, was advocated as a means of surgical treatment. Bier, however, by intermittent compression hyperemia by a neck band, thereby causing a variation of cerebrospinal fluid pressure, was not able to produce attacks in epileptics. He therefore refutes the Kocher hypothesis. Former investigations by the author in conjunction with Pötzl, of fluid pressure measurements in a considerable number of nontraumatic epilepsies, has not confirmed the contention of Kocher, although a certain significance of increased pressure is not denied. In twenty-six epileptic cases of this series lumbar puncture was done. In nine cases the pressure was within normal limits (120-150 mm. water); in eight cases between 150-180 mm.; in five cases between 180-200 mm.; in four cases over 200 mm. One patient, who later showed abscess formation, registered a fluid pressure of 160 mm. In eight cases in which an estimation of the protein content and cells was made, no deductions were made. In the author's opinion, Kocher's contention that a constant and significant increase of fluid pressure is present in these traumatic cases is not proved.

A survey of the German literature of war head injuries shows that considerable attention has been paid to the pressure and state of the cerebrospinal fluid. Tillman attaches considerable importance to general and local increase of fluid pressure as a cause of epileptic attacks. When the increase is in the subarachnoid space, stasis causes autointoxication of the brain by metabolic bye-products. Increase of fluid protein indicates inflammatory reaction.

Bungart believes that all gunshot head injuries are accompanied by a fluid pressure at or above the upper limit of normal. Payr associates meneingitis serosa with brain abscess, and thus explains alterations of the fluid. Hahn makes the following deductions: With normal pressure there is no danger of abscess; the brain defect may be covered. Increased pressure, but no protein increase, shows serous meningitis. These patients are prone to later epileptic attacks, and preventive surgical procedures to relieve pressure must be considered in order. Lumbar puncture alone does not permit a differentiation between encephalitis and abscess formation.

Puncture headache, backache and symptoms of meningismus, rigidity of neck and nausea were of frequent occurrence. These symptoms were also found following dry puncture. Two dry punctures were followed by attacks, as were also successful punctures. In a number of cases Bier's experiment of compression hyperemia was repeated with the result that the fluid pressure was somewhat increased, but no epileptic attack was produced.

A comparison of a series of gunshot injuries of the head shows that forty-four patients with epilepsy had splinters of bone or metal in the brain in 33.6 per cent., whereas in thirty-seven cases without epilepsy such splinters were present in 21.6 per cent. The presence of splinters, therefore, would not appear to be a determining factor in the attacks. Adhesions between the brain and its membranes, and the dura and the bone have been considered by many writers of most important pathogenetic moment, the effect being considered as mechanical and dependent, not on movement alone, but on each pulse beat, coughing and straining. When the injury does not affect the motor areas directly, necropsy cases have shown edema, cystic degeneration and inflammation extending to the motor areas as the most probable causes of attacks. It is thought by the writer that only by postoperative observation of the course of a considerable number of cases may the question of adhesion irritation and reflex action be decided.

The interval cases of epilepsy and the conditions that eventually cause attacks after weeks and months and even years after injury, are subjects of discussion. Encephalitis and meningitis epilepsy are compared. Summation of stimuli may be a factor dependent on increasing cicatrization, cystic formation, etc. Cases of head injury not developing epilepsy are finally considered. The author is inclined to believe that these patients differ from the epileptic patients in that the latter show a greater quantitative change in respect to the lesion, as well as a different localization.

The therapy of traumatic epilepsy is essentially surgical, although there is no consensus of opinion as to the indications or time of operation. The early results of operative treatment are always questionable, as one was accustomed to speak formerly of cure in one or two years, then of longer periods, until it is now held by some writers that a cure cannot be claimed within six years following operation.

A number of surgical procedures are described in the literature; excision of the cortical motor centers after Horsley; subcortical incisions of the same areas after Trendelenburg and covering in of the defect. In general, the last procedure has not been markedly successful. The clearing up of the wound by either primary or secondary operation, removal of splinters, opening of cysts and puncturing an edematous arachnoid are, of course, evident indications. The foreground of surgical interest has been occupied by severing and preventing recurrence of adhesions. Toward this end a fascia-flap has been transplanted in the dural defect, as suggested by Rehn. Koennecke has

recently studied the results of this procedure histologically, and warmly recommends its adoption. A number of dura plastic operations have been devised and the method of Finsterer has received especial attention. This consists of reconstructing the subdural and subarachnoid spaces and inserting a piece of prepared hernial sac so that it will extend beneath the edges of the dural defect.

The author's patients were operated on by a number of different surgeons, many of the operations being early ones for the removal of splinters, foreign bodies and for the purpose of removing dead tissue and cleansing the wound. Many late operations for abscess cyst formation, for adhesions and for defects of bone and dura were also performed. In no case was a persisting successful result as regards recurrence of attacks obtained. The same experience has followed drug therapy, luminal and bromids.

The procedure of Spielmeyer—application of cold by means of an icebag for one-half hour twice daily over the lesion—has been tried by Podmaniky without success.

The article is a comprehensive one of 110 pages, the literature references are numerous both in text and bibliography. The case histories of thirteen cases with illustrations follow the main text. The article may be considered representative of the experience of the central allies in traumatic epilepsy.

SCHALLER, San Francisco.

PROGRESSIVE SUBACUTE FOCAL LEUKO-ENCEPHALITIS. HENRI CLAUDE and JEAN LHERMITTE, L'Encephale **15**:89, 1920.

The authors borrowed the term leuko-encephalitis from Rémond (of Metz) who used it to explain the pathogenesis of a systematic delirium which he thought was due to the destruction of certain fibers of the white matter of the cerebral hemispheres. His conception was hypothetic and not confirmed by an anatomic examination.

Claude and Lhermitte have applied the term leuko-encephalitis to a disease which they have been able to diagnose prior to death and which is caused by disseminated foci of encephalitis in the white substance of the hemispheres and the corpus callosum, involving also the central nuclei but in no way involving the cortex. These foci develop subacutely and are characterized by alterations of anatomic elements which are quite distinct from those observed in acute encephalitis, the softenings, the scleroses of the multiple sclerosis type, the scleroses of syphilis or the encephalitis of infancy. Finally, the authors state that the progress of the disease is sufficiently characteristic to warrant considering it an anatomic and clinical entity.

The description of a new disease entity is based on a long clinical and pathologic report which the authors summarize thus:

A young man of 20 was admitted to a hospital for an excoriation of the foot due to irritation by his shoe. He was without any appreciable hereditary or acquired disease. He began to suffer at the time of admission, i. e., in the month of August, 1915, from weakness of the lower extremities. This increased so that at the end of three months it was quite impossible for him to walk. This paraplegia, which was accompanied by complete absence of the tendon reflexes and by marked signs of incoordination, soon diminished. Then it became worse so that the patient was completely bedridden on Dec. 15, 1915. At this time the clinical picture was quite different from that of October for there was present a marked contracture of the lower extremities,

and an almost complete paralysis of the upper extremities. Moreover, the patient's mental state, normal until then, became abnormal in that he sank into a stupor and was incapable of response. Finally, the sphincter control was lost.

Just after this had occurred, a marked improvement began. He emerged from the stupor on Jan. 10, 1916; voluntary movement returned in all the extremities, and in February the patient was normal mentally, was able to get up, walk and execute all movements of the upper extremities. A rapid convalescence was expected. This expectation was not realized as on April 20, 1916, fever appeared at the same time as a return of the paralysis of the extremities and the patient's speech became so confused that it was incomprehensible.

On May 15 there was great improvement. The patient was able to stand up and walk with the aid of two canes and two nurses. The movements of the lower extremities were poorly coordinated. A month later (June) a complicated group of symptoms was observed which included aphasic phenomena, that is, alexia, agraphia and apraxia. The examination showed that speech disorders as well as those of voluntary movement (apraxia) were completely independent of all sensory disorders of function. Not only was no anesthesia demonstrated but examination of the fundi was completely negative.

Except for the appearance of vomiting of cerebral type and of some unsystematized delirious ideas, the patient's state remained about the same until his sudden death January 10. The authors add that, although there was no further improvement as marked as that recorded above, nevertheless the psychic and aphasic manifestations showed remarkable oscillations. The spinal fluid was negative in every way.

The alteration of periods of aggravation and improvement constitute one of the most striking features of the case above summarized. It was due to this special form of evolution and to the absence of all symptoms signifying a cranial hypertension (absence of choked disk and of increased pressure of the spinal fluid measured by Claude's manometer) that the authors were able to reject the hypothesis of brain tumor and to make the diagnosis of central encephalitis, involving the centrum ovale and the corpus callosum. The anatomic examination proved that this diagnosis was correct.

The necropsy findings summarized were: There were lesions of the centrum ovale, corpus striatum and optic thalamus. The cerebellum, spinal cord, medulla, pons and midbrain were negative except for secondary degeneration of the pyramidal tract. The cortex was normal. The lesions were greatest in the centrum ovale and radiated toward the gray covering on one hand, toward the basal ganglions on the other. The lenticular nucleus and the optic thalamus were most affected. The lesions were limited by the ependymal epithelium, below which the ventricular neuroglia proliferated abundantly. In the centrum ovale, the lesions extended to the corpus callosum and the white commissure and the lesion was continuous from one side to the other. Neither the meninges nor the blood vessels were diseased.

The minute changes were of two types, inflammatory and necrotic. The former were shown by round cell infiltration in the perivascular space and in the walls of the vessels. The neuroglia was greatly increased. There were astrocytes and large giant cells with thick processes. The latter, the necrosis, showed as distinct destruction of small portions of the cerebral parenchyma in

spots of varying size. The vascular lesions were not all of the same age. In some places the round cell infiltration was most conspicuous, while in others the process was older, there being vascular sclerosis and hyaline degeneration. In some vessels obstructing thrombi were seen. All intermediary stages were found. This thrombotic process accounts for the areas of necrosis described.

The authors believe the process is of toxic infective origin and characterized by a vascular inflammatory reaction leading to thrombosis which in turn leads to necrosis of the various parts of the brain mentioned above.

They review the literature briefly and state that but one case corresponds to this one, namely, a case reported by H. Claude and P. Lejonne. In this case no necropsy report confirmed the diagnosis.

Claude and Lhermitte emphasized particularly the remissions as forming a prominent characteristic of the disease.

KRAUS, New York.

STUDY OF ADMISSIONS TO SING SING. BERNARD GLUECK, Ment. Hyg. **2**:85 (Jan.) 1918.

Criminology should seek in psychiatry an end toward the definition and administration of its problem because:

1. Of 608 adult prisoners studied by psychiatric method, 66.8 per cent. were not merely prisoners but persons who had shown through life a tendency to behave in a manner at variance with the behavior of the average normal person, and this deviation from normal behavior had repeatedly manifested itself in a criminal act.

2. Of the same series of 608 cases, 359 or 59 per cent. were classifiable in terms of deviations from average normal mental health.

3. Of these 359 deviations, 171 or 28.1 per cent. of the 608 persons studied possessed a degree of intelligence equivalent to that of the average American child of 12 years or under; of ninety-eight native born defectives, 80.6 per cent. were recidivists in crime.

4. Of the 359 cases, 115 or 18.9 per cent. of the 608 were constitutionally inferior or psychopathic.

5. Of the 359 cases, 73 or 12 per cent. of the 608 were suffering from distinct mental diseases or deteriorations.

It is inferred from these fact that one is dealing with a highly selected and highly specialized group of human beings and to the student of behavior a knowledge of the person back of a given act is absolutely essential if a clear understanding of the nature of behavior is to be had. No agency concerned in the administration of this problem must lose sight of the common goal or end, namely, the necessity for readjustment of that badly adjusted person, the criminal; but in emphasizing the constitutional factors, the psychiatrist must not ignore many environmental factors which contribute directly or indirectly to commission of crime.

These criminals may be divided into the following classes:

Classification.—(1) intellectually defective, (2) mentally diseased or deteriorated, (3) psychopathic, (4) unclassified (epileptic).

General Statistical Considerations.—Of 608 cases, 359 or 59 per cent. were classified as follows:

	Number.	Per cent.
Mentally diseased or deteriorated	73	12
Intellectually defective	171	28.1
Psychopathic or constitutionally inferior	115	18.9
	359	59
Type of offense:		
Crimes of acquisitiveness	388	63.8
Crimes of pugnacity	148	24.3
Sex crimes	60	9.9
Perjury, dynamiting, arson, aiding escape of prisoner, abandonment	12	2
	608	100.0

Thus, frequency of a given type of offense is in inverse ratio to its seriousness, and extent of psychopathologically classifiable cases is in direct ratio to the seriousness of the offense, thus:

Acquisitive crimes psychopathologically classifiable	57.2
Crimes of pugnacity psychopathologically classifiable	58.8
Sex offenses psychopathologically classifiable	70.0

Recidivism.—There are two methods in estimating the degree: (a) frequency with which the individual had come in contact with the law; (b) history of previous sentences to penal or reformatory institutions. In this study the latter method is used. Of 608 cases, 406 or 66.8 per cent. had served sentences before. The factor of nativity was extremely significant, for in the foreign born, recidivism was 49.8 per cent.; in the native born, recidivism was 75.9 per cent.

This decidedly lower percentage of recidivism in the foreign born is undoubtedly due to the fact that accidentally environmental factors play a much greater rôle in the foreigners' conflict with the law.

1. *The Defective Group.*—171 cases or 28.1 per cent. of the 608 cases studied were diagnosed as intellectually defective. In arriving at the diagnosis of mental deficiency, the current psychometric methods were used, Yerkes-Bridges point scale, Terman revision of Binet-Simon test, and the Healy construction test; the diagnosis does not rest solely on laboratory findings, but takes into account also the actual capacity for adaptation which each person manifested in his past life.

Notwithstanding the urgency to do everything possible to readjust the adult offender at the time of his contact with a state prison, the time for active effort in the majority of instances was in the past, especially during their contact with public schools.

The 171 cases comprised all of the defectives among the 608 cases, but as sources of information in cases of foreign born were incomplete, the detailed consideration of the problem of defectiveness will be confined to native born, ninety-eight cases or 57.3 per cent. of the defective group. Of these ninety-eight, the majority fall in various grades of moronism. These findings are particularly significant, as they demonstrate that the higher grade of defective constitutes the most serious problem in antisocial behavior.

Recidivism: Under this head were included seventy-nine cases out of ninety-eight, or 80.6 per cent. Compared with the general percentage in 608 cases, 66.8 per cent., we have an increase of 13.8 per cent., a rather significant correlation between mental defect and tendency to antisocial behavior.

Heredity: Hereditary factors of some significance came to light in forty-eight of ninety-eight cases—principally alcoholism and tuberculosis.

There were early pathologic manifestations in fifty-two instances, such as running away from home, delayed nocturnal enuresis, stuttering, sleep walking, early criminality, hysterical seizures and convulsions. These manifestations might have been noticed had the persons been reared in the proper environment. In twenty-five cases evidence came to light of association in early life with criminal and antisocial types, and in many instances they were deprived of parental supervision. Failure to appreciate these indications of a pathologic state during the school career cannot be easily condoned. At that time some provision should be made for placing these persons in a more appropriate environment.

2. *Mentally Diseased or Deteriorated Group.*—Seventy-three cases or 12 per cent. of the 608 were classified as follows: arteriosclerosis, 3; morphin, 1; dementia praecox, 36; manic-depressive psychosis, 2; markedly cyclothemic, 1; paranoid state, 3; syphilitic, 13; alcoholic, 14. Total, 73.

Recidivism: Of seventy-three, forty-six or 63 per cent. were recidivists. As has been stated, 406 or 66.8 per cent. of the 608 were recidivists. Therefore, as far as mental disease is concerned, the problem of habitual recourse to antisocial behavior is not an especially significant one as compared with the defective and the psychopath.

3. *The Psychopathic or Constitutionally Inferior Person.*—One hundred and fifteen or 18.9 per cent. of the 608 criminals were thus classified. Diagnosis was based on a study of the life history of the person and his mode of reaction to various environmental influences. In addition, certain laboratory tests were carried out which aided in demonstrating the high degree of suggestibility and emotional instability that are so characteristic of the psychopath. Here again only native born are considered. Of 115 cases, ninety-one or 79.1 per cent. were native born.

Recidivism: Of ninety-one, seventy-nine or 86.8 per cent. were recidivists, a higher percentage than in any other group and 20 per cent. higher than in the 608 cases; therefore, the psychopath has contributed in the largest measure to this group of *habitually* antisocial people. For the seventy-nine recidivists in this group the average number of sentences per man is 3.9, although the most requent chronologic age in the 608 cases was only 23 years.

Foreign Group.—Of 608 cases, 213 or 35 per cent. were foreign born, Italy and Russia contributing the most significant share. The average foreigner was committed at a later age than the native born—the foreigner at 27 years, the native at 22.

Recidivism: Only 49.8 per cent of foreigners came under this head as compared to 75.9 per cent in the native born. Foreigners may be called "accidental offenders," adverse social and economic conditions frequently adding to the chances of committing an offense.

In estimating intelligence in the foreign born factors of education, length of residence in the United States, age at arrival, and language factor involved in intelligence scale were taken into consideration.

Italian Group: This comprised sixty-eight cases. Forty-three persons or 63 per cent. were mental anomalies. The high percentage of illiteracy and meager education of those who did attend school, accounts in a measure for the general low level of intelligence as shown by the intelligence tests. Crimes against person (other than sex), which derived their impulse from the instinct of pugnacity, constituted the largest proportion of all offenses, 48.6 per cent. The economic level was low; recidivism: 22 cases, 32.4 per cent.

Russian Group: Sixty-eight per cent. were Jews. There were fifty-eight cases—thirty-one mental anomalies, or 53.4 per cent; recidivism, 66.3 per cent. Most frequent crimes were those having impulse in instinct of acquisitiveness. Level of intelligence was higher than in the Italian group; but the percentage of recidivism was doubled; with this increase in recidivism was found a more or less corresponding increase in unemployment and idleness at the time of arrest.

German Group: This comprised twenty-five cases. Eight or 32 per cent. were mental anomalies. Recidivism was high—68 per cent. In this matter it is hard to draw any general conclusions, for this group shows on the one hand the lowest percentage of psychopathologically classifiable cases, and on the other hand represents perhaps the group of highest culture. There was a predominant tendency to crimes of acquisitiveness.

Austrian Group: This includes eighteen cases—thirteen or 72 per cent. were mental anomalies—the largest percentage of all; recidivists, 55 per cent.

Miscellaneous Group: (Irish, Greeks, English, French, Dutch, Danish, etc.): This included forty-four cases. Twenty-nine or 66 per cent. were mental anomalies; twenty-two or 50 per cent. were recidivists.

General Conclusions from Study or Foreign Group: 1. Of 608 cases, 213 were foreigners, 35.3 per cent. In the counties from which Sing Sing derives its prisoners, there were 52.2 per cent. more foreigners than native born.

2. Of 213 cases, 124 or 58.2 per cent. were mental anomalies. This is significant when we remember that the majority of these cases were excludable at the time of their arrival in the United States.

3. The immigrant is less likely to commit crime than native born. Recidivism is 49.8 per cent., compared to 75.9 in natives.

4. Immigrants showing higher level of intelligence tend to crimes of acquisitive nature; those of lower levels are guilty of crimes against person and sex crimes.

5. Under the new Immigration Law, forty-four of 213 would have been excluded on account of illiteracy, and thirty-five others on account of coming in contact with the law within five years before arrival in the United States.

6. There is a certain correlation between the degree of recidivism and the extent of unemployment at the time of the commission of the crime.

7. There is no correlation between degree of psychopathologically classifiable cases and degree of recidivism in immigrant groups.

8. There is a slight tendency to naturalization.

Activities of the Clinic.—(a) The clinic should make a delineation of the debits in the prison's make-up which might have been responsible for bringing him in contact with the law. (b) It should outline the type of credit, or the desirable qualities the person may possess, the cultivation or development of which will tend to balance debit side. For this purpose the following plan suggests itself:

There should be a new prison at Sing Sing to serve as a classification and research station for the entire prison system of the state. It is intended to keep each prisoner under close observation and study and under intensive vocational training for such a period to define clearly the problem which he presents—perhaps for three or four months. The problem of the adult prisoner is concerned, then, with five more or less well defined types of persons which the various penal institutions of the state should receive on their discharge from the clearing house.

1. Normal young adults capable of learning a trade should be sent to either of two industrial prisons—Dannemora or Auburn.

2. Older normal prisoners should be sent to the agricultural prison at Great Meadows.

3. Insane delinquents should be sent for permanent care to Dannemora State Hospital; for transitory disturbances, to a psychiatric pavilion at a reception prison.

4. The defective delinquent should be sent to an institution for more or less permament segregation.

5. The psychopathic delinquent—treatment should be determined after a thorough investigation by a competent Board of Inquiry.

SCHALLER, San Francisco.

LOCAL PARALYSIS FOLLOWING SUPERFICIAL INJURIES NOT INVOLVING NERVE TRUNKS; WITH SPECIAL REFERENCE TO TRAUMATIC "ASCENDING NEURITIS" AND TO "REFLEX PARALYSIS." F. M. R. WALSHE, Brain **42**:339, 1920.

The author discusses a group of cases of local paralysis, following trivial superficial injury to the distal extremity of the limb, in which no nerve trunk was involved yet which presented the symptoms of an organic nerve lesion. He suggests two causes to explain the symptoms: (1) "reflex paralysis" and (2) "ascending neuritis." The concept of reflex paralysis is now almost abandoned and is accepted only in cases of arthrogenic paralysis, such as have been reported by Oppenheim and Kramer. He suggests that the ill defined and heterogeneous collection of clinical forms that Babinski has named "reflex nervous disorders" is not a true clinical entity but rather a medley of abnormal conditions to which hysteria and mechanical effects of tissue changes and even local tetanus may contribute their share. He then discusses the alternative explanation, ascending neuritis, which Sicard has recently described in a most comprehensive study. The latter regards the condition as exceedingly rare. He defines it as a localized, slowly progressive lesion, ascending from the distal terminations of the nerve toward a central end. It is constantly associated with an infection of a cutaneous lesion in which nerve endings are affected. In a careful review of the clinical and pathologic studies of French and German literature, he finds that obscurity pervades the whole subject. He states, however, that there is sufficient clinical evidence that the syndrome of "ascending neuritis" is not an example of a "lymphogenous toxi-infective" process as described by Orr and Rows. Four cases presented by the author correspond in their clinical picture to the syndrome of ascending neuritis:

CASE 1.—This consisted of a small suppurating wound of the dorsum of the foot followed by paralysis of all muscles supplied by the external popliteal division of the sciatic nerve with corresponding sensory loss.

CASE 2.—A superficial wound of the lower third of the thigh was followed by paresis and wasting of the quadriceps extensor femoris and diminution of the knee-jerk.

CASE 3.—The patient received a penetrating wound of the leg in its distal third which resulted in drop-foot and diminished faradic excitability of all muscles below the knees. There was no sensory loss; but there were diminished ankle-jerk, cyanosis, coldness and lesions resembling chilblains on the foot.

CASE 4.—A blow received on the heel caused no wound. Severe and progressive atrophic flaccid palsy of all muscles supplied by the sciatic nerve followed with a corresponding change in the reflexes, and sensory loss. There were marked cyanosis and coldness of the foot.

The septic factor is excluded. In conclusion he states that in the absence of any sound pathology of these clinical conditions it is impossible to dogmatize as to the underlying lesion. They indicate, however, that in rare instances the clinical manifestations of a local neuritis may follow trivial injuries commonly associated with organismal infection of the afferent terminations of the nerve affected.

BLITZSTEN, Chicago.

THE SOMATIC CAUSES OF PSYCHONEUROSES. CHARLES L. DANA, J. A. M. A. **74**:1139 (April 24) 1920.

Dana deplores the emphasis placed on the psyche as the sole etiologic factor in the psychoneuroses. His argument is based on the theory of "synapsis," which implies "breakable points and points of variable resistance in the finer organizations of the brain." Here the traveling nerve impulse meets its highest resistance and neural phenomena are apt to develop. Paths of conduction may be blocked or made free not only by mental but also by physical agencies and commotional changes, endocrine disorders, toxemias of various kinds, etc., may all act as etiologic agents. A second hypothesis is presented, and the effect of concussion, as an instance, is traced through the stages of physical changes in the nervous system conditioned by molecular changes, the emotion, the subconscious desires with defense and adjustment activities, the organic or biochemical nerve disturbance and finally the fully developed psychoneurotic phenomenon. It is asserted that the steps of emotion and subconscious desires may be absent altogether and that in nontraumatic neuroses the affect reaction and the organic or biochemical processes are dominant. In any event, there is actual material change in the nervous system. These neural modifications are, of course, temporary and may be adjusted by a mental effort or suggestion treatment.

Dana is theorizing, but his theories are ingenious and interesting and do not wholly lack scientific support. For instance, Carver and Dinsley's experimental research (Carver, Alfred, and Dinsley, A.: Some Biological Effects Due to High Explosives, *Brain* **42**:113 [June] 1919) showed paralyses and nervous disturbances in the fish and rats placed in the zones of indirect concussion. The same observations were made on men working in demolition stations protected from direct concussion and presumably not subject to fear.

It has been the reviewer's experience, that almost 50 per cent. of the cases diagnosed as psychoneuroses of various types, and presenting the symptomatic picture of so-called functional neuroses, revealed on careful and extended physical examination the presence of such conditions as anemia, arteriosclerosis, diabetes, endocrine disorders, nephritis, pelvic disease, pyorrhea, apical

abscesses, syphilis, tuberculosis, etc. It may be too much to assume a direct causal relationship to the neurosis in every instance, yet such definite pathologic states cannot be ignored. An energetic effort to correct these underlying physical defects will often result in recovery from, or marked improvement in, the neurosis.

STRECKER, Philadelphia.

CONCERNING PRISONERS. BERNARD GLUECK, Ment. Hyg. **2**:117 (April) 1918.

There is today a serious lack of coordination of effort in criminal administration. The notion of "born criminal" is fatalistic in its finality. The statement that persons by the very act of their recidivism have given proof of being irreformable, either as born or instinctive criminals or as otherwise predestined to their careers, can no longer satisfy the mind of any one who approaches the situation without bias. As soon as the person is put in confinement he is despaired of, his past ceases to be of interest, his future ceases to be viewed as a problem containing unknown possibilities. It is precisely at this stage that the need arises for close observation and open-minded study of his behavior both past and present if treatment and eventual discharge back into the community is to have any helphul bearing on his future course.

The danger lies in ignoring the medical definition of the man back of the act. There is a fallacy in trying to fit the sentence to the criminal act instead of to the criminal back of the act. The man whose constitutional disease expresses itself in antisocial behavior on the one hand and in disordered mentality on the other, has not received adequate attention when the one phase of his difficulty is dealt with independently by the imposition of a definite sentence, and the other phase by temporary treatment in a hospital for the insane for the period of the acute manifestation of his mental disorder. In either case treatment has been directed to an attribute of his diseased personality and not to the personality as a whole in its relation to environment. The majority of psychopathic persons show in childhood and early boyhood unmistakable signs of pathologic personality. It is in the early formative period, especially during boys' contact with the school system, that a better public appreciation of these danger signs is needed and a disposition to institute the proper preventive measures; there is also necessity for recognition that there is a great problem in understanding the causes and nature of delinquency; there is a necessity for a revision of procedure in the direction of individualization. The community must know: 1. That the surest protection against the spread of crime is effective handling of the criminal. 2. That such knowledge must proceed from a detailed knowledge not merely of his guilt in a given instance, but of himself, his history, his fixed limitations and his possibilities for rehabilitation. 3. That the test for efficiency in a modern penal system is its success in turning out not model prisoners, but decent citizens.

SCHALLER, San Francisco.

NOTE SUR LES CANALICULES INTRACELLULAIRES DES CELLULES NERVEUSES (NOTE ON THE INTRACELLULAR CANALICULI OF NERVE CELLS). J. CARAMANIS, Schweiz. Arch. f. Neurol. u. Psychiat. **5**:264, 1919.

Nelis, Holmgren and Joris have shown that the protoplasm of nervous cells is traversed by delicate canals, which occasionally form a sort of network, to which Nelis gave the name spireme, and Holmgren, the name of reseau canaliculaire. This is present in all the cells of the cerebrospinal axis and

stands in intimate relation with the tigroid bodies. Holmgren and Pugnat thought that this network was connected with the pericellular lymphatic spaces; more recently Holmgren has expressed the opinion that they represent a liquefaction consequent to penetration by neuroglia cell processes, the purpose of which is to serve the nutrition of the cells. According to Legendre this hypothesis is reversed, the glia cells in question playing the rôle of phagocytes. According to Golgi, who used the silver method, these canals represent an intracellular structure. Cajal and Holmgren look on the canals and the reticulum as identical structures, while von Bergen endeavors to distinguish the two.

The author employed the toluidin blue stain, according to the Lenhossek technic. These observations were carried on altogether on the motor cells of the anterior horns of an adult cat, which died of starvation at the end of eight days, and which had otherwise been well. The writer believes that the structures under consideration are formed, not by an intracellular network, but by intracellular canals. These canals present markedly varying configurations, have no characteristic localization, occupy both the periphery and center of the cell, proceed in any direction, even through the nucleus, and anastomose in an intricate manner. Some of these canals are seen to open on the surface of the cell. They do not appear to have an independent wall, but are surrounded by granules derived from the chromophil substance. It is striking, indeed, that these canals are in intimate contact with the tigroid bodies, a finding that convinces Caramanis that there is a functional relation between the two. He is of the opinion that these canals are formed at the moment that the protoplasm disintegrates and probably represents a dissolution of the chromophil substance rather than of the spongioplasm. The formation of the canals is, therefore, the result of an accidental elimination and indicates definite cell pathology. The canals represent the initial stage of cavity formation, which may progress to complete cellular disintegration.

WOLTMAN, Rochester, Minn.

VACCINE THERAPY IN CEREBROSPINAL MENINGITIS. LUCIEN GIRARD, Bull. méd. **34**:313 (March 27) 1920.

The proper therapy of cerebrospinal meningitis due to the meningococcus, the author says, is a combination of serum and vaccine therapy; he gives several reasons for this. Serum alone is inefficacious when the infecting germ is atypical and no specific serum is at hand, when the infecting germ is resistant to the serum, when there is a meningococcemia, and when the meningitis has entered on a chronic phase with or without localization of the infection in the middle ear, or elsewhere.

Nine cases are cited (reported by others) in which cure was obtained by vaccine therapy. An autovaccine is presumably not considered superior, though the results thus far reported have been obtained largely with autovaccines only because stock vaccines are not easy to procure. That prepared by Sergeant and Bordet is the only one at present (in France).

These points should be added regarding dosage and administration: Boidin and Weissembach commence by injecting 350 million organisms subcutaneously. Florand and Fiessenger make an initial injection of 1,000 millions, while Mery and L. Girard (the writer) begin with only 200 millions. The doses are gradually increased until 2, 3, 5 or 10 billions are reached. The

injections are given at intervals of three or four days or a week. Recently, Sergeant and Bordet report the intravenous use of a stock vaccine in a meningococcemia (with cure).

Lewkowicz has shown that under the influence of the vaccine the serum of the patient acquires immune properties and the titer for specific antibodies becomes equal to that of the therapeutic serums of commerce.

DAVIS, New York.

OCULAR ATAXIA OF TABETICS. A CANTONNET, Presse méd. **28**:156 (Feb. 25) 1920.

Paralyses and pareses of extrinsic eye muscles in tabes are contrasted with ataxias in a study of cases reported by various observers. These ataxias—disorders of the associated movements of the eyes—are uncommon, but have been found either in the presence or in the absence of ataxia of the extremities.

Ocular ataxia may be evident on the simplest observation, appearing as an irregular and independent stopping and starting of the two eyes when an attempt is made to follow a finger moving from side to side; or it may be latent. A simple means of demonstrating this is quoted from Miraillé and Declaux: A truncated cone of paper 25 cm. long, rolled to 4 cm. in diameter at the proximal end and 1 cm. at the distal, is placed before one eye of the subject, who fixes an object 5 meters distant. If unstable binocular vision is present, diplopia appears when the tube is in place and disappears when it is removed, since the axis of vision of one eye is fixed and the other eye permitted to drift. This procedure shows simple eye muscle palsies as well as incoordination, but a differential diagnosis can be made by observing the direction and degree of diplopia, which are fixed in paralyses and variable in ataxias.

HUDDLESON, New York.

A PROPOS DES GRIFFES (POSITION OF FINGERS FOLLOWING NERVE INJURIES). M. GASTON GIRAUD, Progrès méd. **35**:113 (March 13) 1920.

The writer has contrived a means to express by formula the position of the fingers in the various hand positions found in peripheral nerve injuries. In his scheme each finger is represented by a roman numeral, the thumb being I, in each finger each phalanx is expressed by an arabic numeral, 1 applying to the proximal phalanx, and the capital letters "F" and "E" stand for flexion and extension, respectively. A plus sign occasionally used explains itself. How these terms are to be made into a formula is perhaps clear when it is stated that the writer formulates the attitude of the normal hand thus (IV-V) $F_1F_2F_3$. As a further example, in the case of an ulnar lesion in the forearm, because of the loss of function of the interossei and lumbricals, the resultant attitude requires the formula (IV-V) $E_1F_2F_3$. This much gives the method.

Formulas are given for many situations—for several subgroups under each of his three main classes, which are: (1) atonic hand attitudes; (2) hypertonic hand attitudes, and (3) hand attitudes with contractures.

This sort of work is not lacking in scientific accuracy, but it is academic and hardly of practical benefit. Such a method could not be used from a book by some one not already able to think in terms of the nerves, muscles and joints involved; and, on the other hand, any one so trained would arrive at the diagnosis without the aid of so mechanical a procedure.

DAVIS, New York.

A SIMPLE METHOD OF DISTINGUISHING WHITE FROM RED CELLS IN SPINAL FLUID CELL COUNTS. S. R. GIFFORD, J. A. M. A. **74**:1024 (April 10) 1920.

In the procedure employed by Gifford, the fluid is taken in two test tubes. To one of these, containing about 2 c.c., is at once added one drop of Loeffler's methylene blue, and the tube shaken gently. The second tube is saved for other tests. Examination after two minutes reveals the nuclei of the white cells staining distinctly so that they may be easily distinguished from red cells by low power, and lymphocytes from polymorphonuclears by high power. If the stain does not act quickly, two or more minutes may be required, but never long enough for coagulation to affect the results appreciably, except in a very heavy, turbid fluid.

KLAUDER, Philadelphia.

MYOCLONIC ENCEPHALITIS. J. A. SICARD, Presse méd. **28**:213 (April 14) 1920.

Written Feb. 6, 1920, this contribution takes up five cases in detail, observed during the preceding three months; up to that time no cases of encephalitis lethargica with myoclonic symptoms had been reported (in France), but a note appended to the article cells attention to the large number reported in the interim, from February to April. It is stated that some observers believed the myotonic to be more frequent than the ocular or lethargic manifestations. Transitional cases had also been reported.

The cases described here were characterized by a week or ten days of severe cervical, brachial and intercostal neuralgic pains, with moderate fever, followed by a period of about the same length or less when these pains disappeared and myoclonic contractions set in. During the third week, delirious symptoms were apparent, progressing slowly to coma and exitus; or else, a slow improvement appeared about the third week and recovery took place. The contractions typical of these cases were generally strong, rhythmic, occurring every minute or two, in one or a group of muscles, and showing a predilection for abdominal wall and diaphragm. Other findings were characteristic of encephalitis lethargica as earlier described.

HUDDLESON, New York.

ALIMENTARY ANAPHYLAXIS DUE TO PANCREATIC INSUFFICIENCY. M. NATHAN, Bull. méd. **34**:59 (Jan. 21) 1920.

The writer recounts a case that offered convincing clinical confirmation of the experimental work of Lesne and Dreyfus. These workers have shown that egg albumin subjected to the action of gastric juice, retains its ability to cause an anaphylactic reaction when injected into a sensitized animal, but that the anaphylactic reaction is absent if the egg albumin is subjected to the action of pancreatic juice before injection. His patient was a child of 8 years who had always shown marked (intestinal and skin) anaphylactic reactions to ingestion of egg albumin in any form. The child was normally developed and examination of the abdomen showed only a slight distention. However, analysis of the feces showed pancreatic deficiency (numerous undigested meat fibers). The child was given 40 centigrams of pancreatin a day and while taking this, egg was added to the diet successfully. Several times the pancreatin was suspended and immediately there was a return of the

anaphylactic reaction. The latter feature excludes the possibility that the method of giving the egg albumin (in small increasing amounts) had been responsible and makes clear that the pancreatic feeding was the essential thing. The writer believes that every case of anaphylactic reaction to egg albumin should be examined for possible pancreatic deficiency, either by the Cammidge method or by the examination of the stools, and believes that prolonged, perhaps permanent, feeding of pancreatin will be the treatment of choice.

DAVIS, New York.

GENERAL ABSENCE OF TENDON REFLEXES IN CASES WITH WOUNDS OF THE CRANIUM INVOLVING THE MENINGES. A. SOUQUES, Méd. **1**:274, 1920.

Souques believes that this symptom has often been overlooked. He believes it due to pressure on posterior spinal roots by spinal fluid at increased pressure. He found increased pressure and increased albumin in cases of trauma three or four months after the injury. This he believes due to mild infectious meningitis which passed unnoticed, and of which the increased pressure and albumin were the only evidence.

He cautions against mistaking areflexias of this kind in nontraumatic cases for tabes dorsalis.

KRAUS, New York.

PSEUDOHYPERTROPHIA MUSCULARE DE LA JAMBE CONSECUTIVE A UNE BLESSURE DU NERF SCIATIQUE (PSEUDOHYPERTROPHY OF THE LEG FOLLOWING A WOUND OF THE SCIATIC NERVE). L. CORNIL, Progrès méd. **35**:117 (March 13) 1920.

In a case seen twenty-eight months after injury (a buttock wound by a bullet) there was a clinically incomplete interruption of the internal popliteal with slight irritative painful phenomena, fibrillations, impaired (returning) flexion of toes and foot, tactile and pain hypesthesia in the zone of the posterior tibialis, partial reaction of degeneration in the zone of the internal popliteal, abolition of the ankle and medioplantar reflex, and as the striking sign a hypertrophy of the calf muscles on the affected side. Subcutaneous edema and thickening of adipose layers were not present to explain the finding nor were there circulatory modifications. The muscle mass itself gave a marked feeling of elasticity. The author reports the case as one showing "true interstitial muscular pseudohypertrophy" dependent on nerve alterations, and mentions that two similar cases have been reported by Lhermitte and another by Tinel.

DAVIS, New York.

FIFTEEN CASES SHOWING INVOLUNTARY MOVEMENTS FOLLOWING ATTACKS OF GRIP AND LETHARGIC ENCEPHALITIS. PIERRE MARIE and G. LEVY, Méd. **1**:270, 1920.

This is an account of myoclonias of the head and extremities appearing during epidemic encephalitis. It is merely a review of these associated clinical manifestations accompanying the disease. They differ in no way from what is commonly found. This review is interesting.

KRAUS, New York.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 27, 1920

S. D. INGHAM, M.D., *President*

AN UNUSUAL CASE OF OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY. Presented by DR. N. W. WINKELMAN.

A man, aged 54, whose family history was negative, denied syphilis, but admitted that he drank to excess. On Sept. 28, 1918, at 8 a. m., while waiting for a car, he had severe occipital pain, became unsteady, staggered, but reached home without assistance. He could not speak above a whisper and had much difficulty in swallowing. He remained in bed a month because of unsteadiness in walking. The left side of his body felt as though asleep. At present he has a tendency to stagger and heat applied on the left side of the body gives a sensation of tingling.

Physical Examination.—The base of support was widened, and he was unsteady with eyes open or closed. The right pupil was smaller and the right palpebral fissure was narrower than the left; a slight right enophthalmos was noted. There was no nystagmus; the uvula was drawn up and to the left. The motor system was normal.

Sensory Examination.—Sensation of the face was normal. Sensation to touch of the body and extremities was normal. Sensation of pain, heat and cold was markedly lessened on the left upper and lower extremities and the left half of the body. The pharyngeal reflex was absent. All other reflexes were normal. The blood pressure was: systolic, 152; diastolic, 82. The Wassermann and the urine tests were negative. Bárány tests were made. The lesion was located in the pontile region. The peculiarity in this case was the non-involvement of the descending root of the fifth nerve. It is the first case of its kind in the literature.

A CASE OF BRAIN TUMOR WITH WERNICKE'S HEMIPUPILLARY INACTION SIGN. Presented by DR. SHERMAN F. GILPIN for DR. F. X. DERCUM.

J. D., a boy, aged 9, was admitted to the Jefferson Hospital, Feb. 6, 1920. He had been suffering from vomiting, some disturbance of vision and headache.

The family and personal history were negative.

Present Illness.—Three months ago the patient began to vomit and had a slight headache. For two weeks he vomited about every other day; then the vomiting increased to about twenty times a day. Severe temporal headaches accompanied the vomiting. After a vomiting spell, which sometimes lasted from six to eight hours, the patient was hungry and retained food. Spells of such severity occurred every few days.

Just before Christmas the eyes became crossed. This condition has persisted. Two weeks ago, during a spell, he said he could not see and acted queerly. After a spell is over he does not remember anything about it.

He vomited from midnight of February 4 until the following forenoon; he screamed continuously and was difficult to control. February 6 he was apparently all right again, and examination showed slight weakness of the left arm and leg; face normal. There was slight incoordination of the left arm with adiadokocinesis and asynergia of left leg. Gait and posture showed a slight tendency to spread the legs. The reflexes and sensation were normal. There were choked disks with white star-shaped figures in the macular region and retinal hemorrhages. Left homonymous hemianopsia was readily demonstrated. The left pupil was larger; there was paresis of the left external rectus and extreme rotation to the left caused nystagmus. The pupillary reactions were repeatedly studied by Drs. Dercum and Gilpin. The left pupil was absolutely immobile to light, the right pupil failed to respond so long as the beam of light was thrown on the blind half of the retina; it responded by contraction at once when the beam of light was thrown on the side in which vision was preserved. A small pocket-light throwing a narrow beam was used. After about ten days the response became markedly less and finally disappeared so that the right pupil, like the left, became fixed to light.

The tumor was probably so situated as to give rise in a moderate degree to cerebellar symptoms and at the same time so far forward as to involve the optic tracts in relation with the primary optic centers or possibly these centers themselves. Dr. Dercum has seen the Wernicke sign four times and in one instance the clinical report was accompanied by necropsy findings. (See "Tumor of the Thalamus, More Especially of the Pulvinar, Presenting Wernicke's Pupil Reaction," *J. Nerv. & Ment. Dis.*, August, 1890.)

DISCUSSION

Dr. Charles K. Mills believed that this sign was important and capable of demonstration, although Philadelphia and New York ophthalmologists have denied the possibility of demonstrating it.

Dr. Mills was struck with the similarity between the case reported by Drs. Gilpin and Dercum and the first of several cases included in the paper of Dr. Seguin in the *Journal of Nervous and Mental Diseases*, volume 14, November and December, 1887, pp. 721-737, entitled "A Second Clinical Study of Hemianopsia; Cases of Chiasm-Lesion; Demonstration of Hemiopic Pupillary Inaction." In the case presented, the most important features are that the patient has almost entirely lost sight in the left eye and in the right eye has considerable impairment. This loss and impairment are apparently due to atrophy consecutive to a papilledema. The other symptoms point to a cerebral growth, and it is altogether probable that this neoplasm is located so as to involve the interior aspects of the right temporal lobe and to produce compression on the optic nerve between the chiasm and the pregeniculum.

In Seguin's first case there was loss of vision in the left eye, temporal hemianopsia with the right eye, complete atrophy of the left optic nerve, and partial atrophy of the right, with no other symptoms of cerebral lesion, and a demonstrable hemiopic pupillary inaction from examination of the right eye.

Dr. Mills believed it impossible to demonstrate the hemiopic pupillary inaction in the case presented as the methods adopted were not correct. It was of no avail to flash the light of an electrical ophthalmoscope to the right and left without closely restricting the illumination to one-half or one portion of each side of the retina. In order that this sign may be demonstrated it is absolutely necessary to take certain precautions and carry out a certain method.

He had on several occasions had this Wernicke pupillary inaction demonstrated for him by Dr. S. D. Risley and years ago he had been able with great difficulty to demonstrate the sign himself.

Dr. Langdon said that the best instrument at present was an instrument used for the transillumination of retinal detachments. It was a complete metal globe containing an electric light bulb, the globe having an opening on one side which was very small, and which projected a narrow beam of light; by the use of this the light could be thrown on any part of the retina desired, and then the only possibility of other parts being illuminated was by diffusion, which it seemed was impossible to be sure was overcome. At the University Hospital this has been tried on many cases of hemianopsia but without eliciting Wernicke's sign. Many ophthalmologists are skeptical concerning the possibility of showing the presence of this sign.

Dr. Gilpin said that the fact that this had gone on for four months, that he had such a marked choked disk, that he had some difficulty in coordination which showed more markedly than it did at the present time, led him to suspect that the lesion was within the cerebellar fossa and by interfering with the flow of the cerebrospinal fluid had filled up the lateral ventricle. He believed in some way pressure had been relieved so as to allow freer circulation. The boy was better than when he came in.

Dr. Gilpin said he knew that ophthalmologists take no stock in Wernicke's sign, but he was sure that in this case they had obtained it.

A CASE OF EXTRAPYRAMIDAL LESION. Presented by Dr. A. E. Taft.

E. Y., aged 9, had an unimportant family and personal history. She complained of weakness of the left side. Her left foot had always been weak but more so of late. During the past few months she had not used her left upper limb.

Examination.—There was some weakness of the lower part of the left side of the face. She showed her teeth with good bilateral innervation, but on elevating the corners of her mouth separately, it was done less well on the left than on the right side. The pupils were normal, and the tongue was protruded normally. The left upper limb was slightly smaller than the right. It was spastic and there was increase of tonicity both on active and passive movement. This was not constant in degree. Spasm of the limb prevented eliciting the tendon reflexes on this side. Those on the right side were normal. There were transient involuntary movements of the fingers, and occasionally of the forearms. These movements suggested beginning athetosis. The grasp of the left hand was good but weaker than the right. There was no disturbance of general sensation, stereognosis nor position sense. Lower Extremities: The left foot was turned in. The left lower limb was spastic; the right was normal. Tendon reflexes were somewhat exaggerated on the left, but not on the right. There was no sensory change, and no ankle clonus, but there was a Babinski reflex on the left. It is of special interest that the weakness of the left upper limb has been noted recently, while that of the lower limb was noticed when walking began.

DISCUSSION

Dr. Charles K. Mills said that the case was of great interest to him with regard to the existence, position and characteristics of the cerebral tonectic apparatus. This he believed had both a cortical and a lenticular representa-

tion. The cortical rendezvous of tone is probably in the midfrontal region. Dr. Mills noticed, in looking at this girl and also in testing her grip, that she probably had a species of preservation, although this was not marked. She had athetoid movements and did not seem to let go as quickly with the left hand as with the other. Therefore he thought that the diagnosis which had been intimated was correct, namely, an extrapyramidal, probably a premotor cortical or ganglionic subcortical, lesion.

A CASE OF BRAIN TUMOR OR ENCEPHALITIS ACCOMPANIED WITH DYSPITUITARISM AND SHOWING LONG CONTINUED PSEUDOSTUPOR. Presented by Dr. Charles W. Burr.

Casual observation of the patient would give one the impression that he was unconscious; looking more closely the judgment would be apathy. He lay motionless, staring into space, paying no apparent attention to what was going on around him, responding only to painful stimuli. It was impossible to determine his real condition of consciousness because he was speechless, not from word deafness but because of mental dulness; he also had some defect of vision and was palsied on the left side. He was not in coma, but it is doubtful whether, when not irritated by external stimuli, he carried on any mental process or had any emotional tone. For want of a better name, Dr. Burr has called the condition of consciousness pseudostupor. At times when repeatedly spoken to he responded with a few words. He had been in this condition, not common in brain tumor, at least eight weeks. Dr. Burr had seen profound stupor, so deep that no stimulus caused evidence of consciousness, endure for four weeks in a patient with tumor of the left prefrontal lobe.

The patient was entirely too girlish in appearance for a boy of his years. He probably had some pituitary gland trouble, whether congenital or the result of inflammatory cerebral disease starting in 1918 is disputable.

The patient was 11 years old. The family and personal history was unimportant. The patient had had no serious illness until the onset of the present trouble in December, 1918, with a mild attack of influenza. Soon after he was up and about, his mother noticed that when he reached for an object with his left hand, his arm would fall slowly to his side. This condition grew worse and at the end of three weeks he was unable to lift the arm. He dragged the left leg in walking. At the beginning and for some time after, he complained of intermittent, aching pains, and numbness in the left arm and hand, left side of the chest and left leg. The fixed stare, so noticeable now, dated from December, 1918. This patient was seen by Dr. Spiller on Oct. 29, 1919, who made the following report:

Paralysis of upward and downward movements indicated the lesion to be in or near the corpora quadrigemina. There was no lateral ocular palsy. On looking to the right or left he looked down because of muscular weakness. He would probably have complete ophthalmoplegia finally. He could close the left eye tightly. The corneal reflex was normal. Masseter contraction was good. Weakness of facial muscles on the left side was present. The face was hyperesthetic. He had marked paresis of the left upper limb and marked spasticity of the left lower limb. Left patellar and ankle clonus was marked. There was great exaggeration of tendon reflexes, and a permanent Babinski reflex on the left with a slow Babinski on the right. Pain sensation was acute in both limbs; no loss of sense of position; stereognostic perception good; therefore no involvement of parietal lobe.

Summary: The lesion involved the right side of the brain above the anterior portion of the pons. It was very near and most probably just back of the corpora quadrigemina in the region of the basal ganglions and did not involve the cortex. The lesion was probably a glioma. There was probably no mechanical obstruction of the aqueduct of Sylvius causing internal hydrocephalus. The clinical picture has changed in many ways since Dr. Spiller made the examination.

A later examination by Dr. Burr showed that the pupils were fixed. He was unable to look upward with either eyeball. Horizontal movements were hesitant and jerky, and there were occasionally intermittent simultaneous nystagmoid oscillations of both eyes toward his nose. Rotary oscillation in the upward direction of the left eye was constant in the lateral position of the eyeball. Reflexes were normal on the right side, exaggerated on the left. The Babinski reflex was present on the left side only. Abdominal reflexes were absent. He was weak in the limbs on both sides, much more so on the left, and showed evidence of distinct hemiplegia. Speech was halting, stuttering and unintelligible. There was incontinence of urine and feces. A number of spinal punctures were made, the fluid coming out with great pressure. After one puncture the reflexes were reported almost normal. The cell count was 43 and there were no tubercle bacilli in the smear. The spinal fluid Wassermann test was negative. Roentgen-ray examinations showed the sella turcica to be unaffected, but there was internal hydrocephalus with separation of the sutures of the skull.

On Nov. 1, 1919, ophthalmologic examination showed that the disks were swollen and the veins tortuous; summit, +7; fundus, +5; the disks of the left eye were similar to those of the right but not so marked. Paralysis of upward movement and deficient convergence were present.

Physical Examination.—Examination when admitted to the Philadelphia General Hospital, Jan. 6, 1920, revealed a well nourished white boy. The skin was dry and soft and of rather delicate feminine texture. The contour of the extremities, as well as that of the trunk and pelvis was feminine. The genitalia were under-developed. The hair of the scalp was abundant. There was practically no body hair. There was no evident enlargement of the thyroid or other gland. The head and face were well developed. The patient was able to wrinkle the skin over the forehead equally on both sides. The masseters acted fairly well. His height was normal for his age. There was no mastoid swelling or tenderness.

The pupils measured about 3 mm. each; were fairly regular in outline and reacted sluggishly to light and accommodation. Extra-ocular movements could not be performed. He would not cooperate. He had a peculiar fixed, blank stare. He was able to close the eyelids.

The tongue protruded without tremor or deviation. Movements were imperfectly performed. There was no apparent difficulty in chewing or swallowing. The speech was somewhat thick, slow and inarticulate. (He talked little and only when strongly urged.) He was unable to expose the teeth on the left side properly.

The right upper extremity was freely movable in all directions. Biceps and triceps reflexes were preserved, but slightly active. Sensation to pinpoint and touch was preserved. On account of mental stupor the patient failed to recognize simple objects, such as a penknife and key, when placed in his hand. The left upper extremity was held flexed over the chest and somewhat rigid.

The chest was well developed. The mammae were too well developed and prominent for a boy. Thoracic organs were normal. Head Measurements: occipital protuberance to line of eyebrow, 10½ inches; circumference, 20¾ inches; meatus to meatus, 13¼ inches.

Eye Examination (by Dr. Langdon).—Pupils: 3 mm., the left was possibly a little larger; slight reaction even to concentrated light. The visual axes slightly diverged. Ocular rotations could not be studied. Vision: light perception only. The mediae were clear; disks very pale, especially the left; margins well defined, but with slight blurring; retinal veins about normal. The arteries were small, but without kinking or other changes. There were no other fundus changes; hyperopia 5 D; no cupping of disks.

In Blockley hospital his temperature has usually been 99; occasionally it has risen to 100, and three times, in the evening to 101. Blood examination, Jan. 14, 1920: Blood Wassermann test was negative. (Spinal fluid examination was not made because of possible brain tumor.) Blood culture was negative.

Roentgen-Ray Report, Jan. 14, 1920: There was apparently a thinning of the bones of the frontal region. Further than this no lesion could be demonstrated. Satisfactory stereoscopic plates of sella could not be obtained because the child moved during the examination.

Dr. Burr confessed that he was not sure as to the diagnosis. The boy either had a tumor—and if so, Dr. Burr agreed with Dr. Spiller as to the location—or he was suffering from encephalitis following, and probably the result of, influenza. Influenza was an exciting cause, the last straw, in many cerebral conditions. Dr. Burr said disturbance of consciousness, such as the boy's, was more common in encephalitis than in brain tumor. It was not coma, but mental sluggishness. Dr. Burr thought such a condition indicated a widespread affection of the cerebrum, just as trance in insanity indicates an affection of the whole intellectual cortex and not merely local disease.

A CASE OF CEREBELLAR LESION. Presented by Dr. Alfred Gordon.

J. O., a boy, aged 13, had been complaining of severe headache for the last two years. During the last six months he had had many attacks of vomiting without any indication of gastro-intestinal disorder. Vertigo began early and is still present but not continuous.

Examination.—When he turned his eyes sharply to the left, nystagmic movements appeared. Occasional and slight nystagmus was present when the eyes turned upward. Both nerve heads were hazy at the margins. There was some optic atrophy and the retinal veins were decidedly tortuous. The left temporal field of vision was diminished. Pain sense was diminished over the entire right side of the face. Hearing was impaired on the right. Reflexes, station and gait were normal. Pastpointing to the right was observed repeatedly in the right hand, but not in the left. A Bárány test performed by Dr. L. Fisher revealed: On turning the test to the right the duration was 38 seconds, to the left, 35 seconds. With the caloric test a douche on the right produced nystagmus of 50 seconds, on the left, of 70 seconds.

In view of the right pastpointing and the localization of the nystagmus, of the findings with the Bárány test, also of the eyegrounds, of the headache and vomiting, the presumption is in favor of a cerebellar lesion situated on the right side.

The ethmoids on both sides were badly diseased, and there was a profuse discharge of pus from both nares. The question arises as to the possibility

of a purulent focus in the right cerebellar region. On the other hand, intracranial syphilis cannot be entirely eliminated in view of the fact that only the blood was examined for a Wassermann reaction, with negative results.

A CASE OF "PURE ALEXIA." Presented by Dr. Alfred Gordon.

A man, aged 72, right-handed, had emphysema of the lungs and endomyocarditis with arrhythmia; arteriosclerosis was pronounced. The blood pressure was: systolic, 190; diastolic, 120. The urine contained a small amount of albumin. The Wassermann test was negative and the eyegrounds normal. Several years ago he had had a mild attack of right hemiplegia from which he made a complete recovery. About one year ago he had an attack of vertigo with severe headache, following which he developed the present condition. He was unable to read printed or written words. Spontaneous speech, repeated speech, singing and understanding of spoken words were correct; reading aloud and mental reading were impossible. The patient was able at times to name some letters correctly, but was utterly unable to make out the sense of the word. Copying was difficult, but writing to dictation and spontaneous writing were almost entirely correct. However, the letters written were much larger than those of the patient's former writing. He could not read what he had written. He recognized an occasional word only when he followed each letter with his finger.

Two other interesting phenomena were present: First, verbal blindness concerned only words, not figures; the patient was able to add, subtract and carry out other arithmetical operations. Second, there was a right homonymous hemianopsia.

The clinical picture of the present case corresponds precisely to the description given by Déjerine in 1892 in which he isolated from Wernicke's sensory aphasia a form of aphasia to which he gave the name "pure word-blindness."

In alexia of the cortical origin due to a lesion of the angular gyrus, we usually observe an impairment of inner language; there is ordinarily a certain degree of word deafness, of paraphrasia and of agraphia for spontaneous writing and for writing on dictation. Moreover, not only words, but also figures are not recognizable by the patient. Finally a homonymous hemianopsia is rarely, if at all, observed in cortical lesions of the angular gyrus.

In the present case the lesion, which is a softening due originally to an embolic occlusion, is probably situated in the inferior longitudinal bundle, namely, fibers connecting the center of general vision with the center of visual images of speech (angular gyrus). Such a conception readily explains the absence of all other aphasic disorders and the presence of the hemianopsia. We are dealing, therefore, with a case of "pure alexia."

THE CONDITION OF A PATIENT IN GOOD HEALTH, BUT EXHIBITING AUTOMATIC MOVEMENTS OF THE LOWER LIMBS, NINETEEN YEARS AFTER COMPLETE TRANSVERSE SECTION OF THE SPINAL CORD BY A GUNSHOT WOUND. Presented by Dr. Williams B. Cadwalader.

The case which Dr. Cadwalader described is the same one that Dr. Francis T. Stewart and Dr. Richard H. Harte reported in the *Philadelphia Medical Journal* of June 7, 1902, under the title of "A Case of Severed Spinal Cord in which Myelorrhaphy was followed by Partial Return of Function."

Their report caused much discussion at the time and the assertion that return of function had occurred in the lower limbs was received with a feeling of skepticism. Notwithstanding the fact that this patient was repeatedly examined by different physicians and was exhibited before medical societies, a definite conclusion was not reached.

Since 1902, such great advances have been made in our knowledge of the physiology of the spinal cord not only through the monumental work of Sherrington in the laboratory, but also by innumerable clinical investigations in man, and most particularly by those of Riddoch, Head and others on cases of complete division of the spinal cord from gunshot wounds observed during the war, that it seemed most important to record the present condition of Dr. Stewart's patient, nineteen years after the injury was inflicted. Dr. Cadwalader knew of no other patient who had survived so long after complete section of the spinal cord. It had been planned to make this report together with Dr. Stewart but he died suddenly three weeks before. Dr. Cadwalader said that Dr. Stewart had agreed with his views.

An abstract of the history as given in 1902 by Dr. Stewart and Dr. Harte follows:

C. N., 26 years old, was admitted to the Pennsylvania Hospital, Jan. 21, 1901, having been shot in the back with a 32 caliber pistol. The bullet had entered 1 inch to the right of the seventh dorsal spine and had passed directly into the spinal canal. There was immediate and complete motor and sensory paralysis below the level of the tenth dorsal spine and all the superficial and deep reflexes were abolished. Three hours after the injury, operation was performed by Dr. Stewart. The laminae of the seventh and eighth dorsal vertebrae were removed, the membranes were found lacerated and the spinal cord completely severed, the distance between the severed ends of the cord being three fourths of an inch. This observation was confirmed by Drs. Mitchell, Fraley and Newlin, who were present. The divided ends of the spinal cord were approximated with catgut sutures and the wound closed. Surgical recovery was uneventful.

After studying the history of this case, it appeared evident that the patient must have been in a state of "spinal shock" on admission to the hospital and that it lasted just three weeks, for it is stated further on in the report that the patellar tendon reflex was detected for the first time on the twenty-first day after the injury. Involuntary movements of the lower limb were noted on the thirty-fourth day. This must have been the beginning of the establishment of the condition called the "mass reflex." From then on the movements observed in the lower limbs could not have been voluntary movements, for at present it can be easily demonstrated that the patient is completely paralyzed in all portions below the costal margins and that the muscular movements formerly regarded as voluntary movements, are in reality the involuntary movements recognized as the "reflexes of defense" or the "automatic" or "reflex" movements that collectively make up what Riddoch has termed the "mass reflex" so essentially characteristic of complete division of the spinal cord.

Examination in 1919-1920.—There was complete paralysis of motion and sensation below the level of the tenth thoracic spine. The lower limbs felt rigid; there was no wasting. The skin over the legs was waxy in appearance and pitted slightly on pressure.

Involuntary flexor movements of the lower limbs occurred at irregular intervals. When sitting quietly, they might be absent for hours at a time, but on the slightest movement of the trunk or on cutaneous stimulation,

marked flexor spasms occurred and if stimulation was rapidly repeated they became violent. The rigidity of the limbs was excessive, requiring considerable force on the part of the examiner to extend the leg but after complete extension had been accomplished, the limb was likely to resume gradually the partially flexed position. This was because of the predominence of the hypertonicity of the flexor muscles. When the patient was prone the limbs could be fully extended by the examiner and they would then remain so until some stimulus caused reflex flexor contraction.

With braces so arranged to extend up to and include part of the trunk and with circular metal strips about the lower leg to prevent complete flexion of the legs, the patient was able to stand erect between two chairs by first supporting herself with her arms and hands and then arranging her lower limbs in extensor spasm favored by gravity, and then removing the support of her arms, only using them to balance with. By this means she could stand erect in a purely "reflex manner" but only momentarily. Tendon reflexes could not be obtained because of rigidity. There was a bilateral Babinski sign, but much of the time the great toe was held in dorsal flexion as a fixed Babinski position. If the soles of the feet were strongly stimulated there followed upward movement of all the toes, dorsiflexion of the foot and strong flexion at the knee and hip.

On scratching the skin of the abdomen there was no muscular response.

Sensation: Sensation of all kinds was abolished. Nevertheless, she complained now and then of a feeling of fulness and discomfort in the abdomen, generally worse after excessive movements of the limbs, and she still had hallucinations as to the position of the toes or feet which were very annoying.

Bladder: The bladder emptied automatically. By practice and judgment she managed to anticipate evacuations and to get along fairly well, but she soils herself occasionally and sometimes wets the bed during sleep.

The patient's general health was good. She was most optimistic and believed that she had recovered voluntary motion. In making a sensory examination, her answers were most misleading. Sometimes when pricking her over the skin of the leg with a pin she seemed to answer correctly, though she did not feel the slightest sensation below the level of the lesion. By long practice she has learned to recognize the traction exerted on sound muscles of the thorax above the level of the lesion caused by the involuntary flexor spasm of the stimulated limb. If it were violent, she occasionally answered that she felt the pin. Moreover, she was so anxious to assist that she unconsciously deceived.

The transitory abdominal discomfort, i. e., a feeling of fulness and distress in the epigastrium and abdomen with nausea that she sometimes complained of, can be explained by assuming that it was the mechanical or reflex result of frequent flexor spasms of the lower limbs and traction on the abdominal muscles. It is significant that the patient stated that she could not vomit. Treatment for the relief of these sensations should be directed toward diminishing the violence and frequence of the involuntary movements by finding the best position for the paralyzed limbs and avoiding undue reflex stimuli. The patient had learned this by experience and fully appreciated its importance. She was cheerful and spent her time sitting comfortably and fully dressed in a wheel chair knitting sweaters, by which means she earned a good income. It must be denied emphatically that return of function had occurred; such a statement was entirely the result of a mistake in observation.

However, the case proves that complete division of the cord at the tenth thoracic segment by gunshot wound is not in itself necessarily fatal, for if the automatic bladder can be established, a patient can live a comfortable chair life indefinitely. Furthermore, it proves that even under the most favorable condition for surgical procedure—for the separated ends of the severed spinal cord were approximated by sutures only three hours after the injury—and after careful nursing for nineteen years, no clinical sign of return of function has appeared; therefore, it can be assumed that regeneration of the spinal cord does not occur, after complete section, and approximation of the ends by suture.

DISCUSSION

Dr. Andrew H. Woods said that the anomalous part of the case as reported was the spasticity in extension in a patient with complete severance of the spinal cord. In the cases reported by Sherrington, Head, Riddoch and others, when there was serious damage to the lateral columns or complete division of the spinal cord, there resulted frequent reflex spasms in flexion defensive in purpose; but there was not the tonic spasticity in extension, which appears to be nature's effort to prevent the body from falling when external disturbing forces have been encountered. The reflex mechanism through which this conservative stiffening of the lower limbs was effected apparently includes an afferent arc which extends from the muscle through the lateral columns of the spinal cord to the region of the midbrain; while the efferent arc according to Walshe possibly runs somewhere near the crossed pyramidal tracts. Hence any serious lesion in the lateral columns of the spinal cord would interrupt this reflex pathway. Dr. Woods thought the explanation given by Dr. Cadwalader of the patient's responses to unfelt sensory stimuli was ingenious and that it seemed adequate.

Dr. Alfred Gordon said that the toe phenomenon in a case of complete section of the cord deserves discussion. The opinion of the majority seemed to be that the toe phenomenon was a cutaneous reflex, dependent on the integrity of the cerebrospinal pathway.

Dr. Cadwalader said that it was true that in cases of complete division of the cord the paraplegia was flexor in type. In this case the spasm of the flexor muscles predominated, but the extensor muscles also showed definite hypertonicity at times. In this respect it did seem to differ from those cases described by Riddoch, Walsh and some other observers, but in spite of this Dr. Cadwalader thought because of the constant predominence of the flexor type of spasm, it should be regarded essentially as a paraplegia in flexion. The "mass reflex" is not so violent or active now as it had been formerly, and he believed this was in part due to the time that had elapsed since the injury, now nineteen years, and in part due to the fact that the patient had learned by experience to avoid all stimuli which provoked reflex action. In reply to Dr. Gordon's question, Dr. Cadwalader said that if the sole of the foot was lightly irritated the big toe would go up in a Babinski attitude, but if the sole of the foot was strongly irritated the toe and whole foot and leg were drawn upward in flexor spasm and would remain in this position some time, but gradually the flexor spasm relaxed and the limbs would lie almost fully extended on the bed. Dr. Cadwalader did not believe that partial preservation of the pyramidal tracts was neessary for such a reflex response.

A SPECIMEN SHOWING EXTENSIVE SUBDURAL HEMORRHAGE.

Presented by Dr. George Wilson.

Dr. Wilson said that his reason for showing the specimen was twofold; first, the long period which elapsed between the accident which produced the subdural hemorrhage and the onset of symptoms; and second, the absence of localizing signs.

History.—The patient from whom the brain was removed was admitted to the Philadelphia General Hospital on Nov. 11, 1919, to the service of Dr. Spiller and died November 15. The man's wife gave the following history:

He was well until four days before admission. He complained of headache on reaching home on the evening of November 6; two days later the headache was so severe that he stayed in bed and called a doctor He was sent to the hospital because of unconsciousness on the morning of November 11. Two months before the patient had had a fainting spell and fell, striking his head on the street car track. Since then he had had an occasional headache which had never interfered with eating, sleeping or working. His wife said that he drank to excess, but as far as she could determine, she was sure that he did not use drugs nor had he ever contracted a venereal disease.

Physical Examination.—The patient was unconscious. The pupils were unequal, the right being larger and did not react to light or in convergence. It was impossible to test for movements of the cranial nerves. There was bilateral choked disk of moderate degree. The deep reflexes were about normal. There was a Babinski sign on the left. There was no rigidity of the neck, no evidence of a fractured skull, and no Kernig's sign. The pulse on admission was 62, regular and of normal tension. The urine, blood and spinal fluid were normal. At necropsy a large, organized, subdural clot was found covering the greater part of the right hemisphere. There was no fracture of the skull. A few years ago Dr. Spiller showed a similar specimen to this society. It is indeed remarkable that a hemorrhage of the size shown here could exist for two months without producing any symptoms outside of an occasional transitory headache.

ON THE OCCURRENCE OF BILATERAL SYMPATHETIC OPHTHALMOPLEGIA IN LETHARGIC ENCEPHALITIS.

Dr. Williams B. Cadwalader.

Dr. Cadwalader thought observation of this symptom exceedingly important because when paralysis of the sympathetic fibers supplying the eyes occurs on both sides it can only be caused by an inflammatory lesion situated in the upper cervical cord, medulla oblongata, pons or perhaps in the cerebral peduncles. The course and situation of the sympathetic fibers to the eye are well known in these portions of the brain, and it is in these parts of the brain that the inflammation is most intense in this disease. Therefore, when associated with the characteristic mental confusion bilateral paralysis of the sympathetic fibers of the eye should be regarded as a diagnostic sign of lethargic encephalitis.

In two cases Dr. Cadwalader said it had occurred early in the course of the disease, but because of the bilaterality it had not been recognized until other signs had developed. Narrowing of the palpebral fissure, myosis and slight retraction of the eyeball occurring on both sides can be readily overlooked whereas if they occur on only one side, they quickly attract attention. The smallness of the pupils in one of Dr. Cadwalader's patients had led to the suspicion of an overdose of morphia.

Recently a case was observed in the University Hospital in which the diagnosis was proved by necropsy and in one of Dr. Cadwalader's cases a postmortem examination had verified the clinical diagnosis.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, March 18, 1920

GEORGE A. WATERMAN, *President Pro Tem.*

DISSOLUTION OF CONTINUITY OF THE SPINAL CORD BY A PROJECTILE PASSING NEAR BUT NOT DAMAGING THE VERTEBRAL COLUMN. DR. H. C. SOLOMON.

Dr. Solomon reported the case of a soldier, 22 years of age, wounded in battle, Nov. 3, 1918. The bullet wound was at the level of the eleventh dorsal vertebra, about a centimeter to the left of the median line. The next day roentgen-ray examination showed a foreign body in the right chest, said to move with respiration. He arrived at a base hospital a week later, at which time he had the symptoms of complete transverse myelitis: paraplegia, incontinence, etc. At this time he already showed several areas of decubitus—over the sacrum and both great toes. He lived until January 10, a little over two months after his injury. During this time there was no change in the paraplegic symptoms, but the decubiti increased.

Necropsy examination on Jan. 11, 1919, showed the body of a markedly emaciated white man, presenting a tremendous bed sore about 7 by 9 inches, extending from the left iliac spine to the right and from the level of the third lumbar vertebra to the anus, with the iliac spines visible and protruding. A small, superficial decubitus was present at the tuberosity of the ischium and decubiti in both popliteal spaces exposed the tibiae and communicated with the joints. There were also pressure sores over the great toes and right heel. Edema was present to the level of the waist. There was a small healed scar at the level of the eleventh rib behind, one-half inch to the left of the spinal column, undoubtedly the point of entrance of the bullet. The bullet track could be traced through the muscles in the lower dorsal region of the back. As far as could be determined, there was no injury to the bones of the spinal column. The foreign body, however, traveled in close proximity to the vertebral column, apparently passing between the transverse processes of the vertebrae. In the middle lobe of the right lung near its hilus, a piece of shrapnel approximately ¼ by ⅛ by ⅛ inch in size was found. The foreign body was walled off with no evidence of suppuration about it.

On removing the dorsal portion of the vertebral column, the dural sheath appeared to be entirely normal. On opening the dura, however, the cord in the dorsal region opposite the bullet track was found to be confluent, consisting of a grayish, liquefied mass. This condition was sharply localized, having a length of 1¼ inches. There was a small portion of the cord posteriorly and laterally that was not entirely liquefied. The pia above and below the injured portion of the cord was discolored a brownish black for an inch or so, apparently an old hemorrhage. The cord above and below this area had its normal shape and consistency. The brain and its envelopes and venous sinuses were grossly normal.

The other positive necropsy findings were: slight pericardial, pleural adhesions, discrete bronchopneumonia of the left lower lobe; slight dilatation of the ureters; cellulitis of the scrotum; acute cloudy swelling of the heart muscle, liver and kidneys and septic degeneration of the spleen.

The cause of the destruction of the spinal cord is not easily seen. Possibly the velocity of the bullet traveling in a closed space may have produced the damage by commotion. A somewhat similar case was reported by Froment (personal communication). In his case the paraplegia had been produced by a bullet passing in close proximity to the spinal column. The paraplegia improved after four or five months. The roentgen-ray findings showed no evidence of fracture.

DISCUSSION

Dr. Henry Viets: In Frazier's book on "Surgery of the Spinal Cord" there is a beautiful illustration of a French case in which the bullet passed near the vertebrae, cracked one of the laminae of the vertebra and caused a complete paraplegia. On necropsy examination no damage to the inside of the dura was found but the cord was completely cut across as sharply as with a knife. This is the only reported case that I have seen.

Dr. W. J. Mixter: I had a rather similar case. A boy was shot with a 22 caliber rifle. He had complete paraplegia. The dura was just nicked by the bullet which did not touch the cord. The cord showed fusiform swelling and its central portion was simply squeezed out. There was practically complete destruction of the cord except for a mere shell.

THE COURSE OF RECOVERY IN SPINAL CORD INJURIES. Presented by Dr. Stanley Cobb.

Dr. Cobb showed a chart on which were plotted the recovery curves of twenty cases of spinal cord injury. These cases were taken as the patients were admitted to Colonel Frazier's surgical service at U. S. Army General Hospital No. 11 at Cape May, N. J. They were studied for from three to nine months. The chart was made by dividing the abscissor into periods of thirty days and plotting against this ordinates in terms of the patient's ability or disability; in other words the heading of the lowest ordinate is "complete quadriplegia"; above this is "paraplegia" and then in order the headings "stands with support," "steps with support," "walks across the room," "walks a mile with support," and "walks a mile"; above this, "well." In this way the general symptomatology is plotted against the number of days since injury and a curve of recovery shown.

The chart showed three main groups of cases: The first group consisted of those whose injury was slight and was discovered only on routine examination. There were three such patients, all of whom showed no disability due to spinal injury at the time of their discharge. The second group was composed of twelve cases and was the most interesting part of the chart. These men showed complete disability after their injury for a period of from fourteen to forty days. They then showed some definite return of function and thereafter rapidly improved for a period of from three to five months. At the end of this second period their recovery curves flattened out and the condition remained practically stationary. The chart, therefore, indicates that in the initial stage of complete paralysis no accurate prognosis can be made, but if no return of function is shown at the end of forty days the prognosis is probably bad. If function begins to return at this time it will proceed for

three or four months, after which there will probably be no more improvement. The third group was composed of five cases which showed no improvement at all from the time of their injury up to the end of the ninth month.

It was attempted to study the bladder, rectal and sexual symptoms in a similar manner, but they were found to be too irregular to chart. It appeared, however, that cervical injuries showed symptoms of this sort only when they were severe. The injuries in the lumbar and sacral regions almost invariably showed these symptoms but in an irregular way; for example, sexual power could be entirely lost and urinary control hardly injured at all, or vice versa.

DISCUSSION

DR. MORTON PRINCE: Some months ago I expressed skepticism regarding the claimed good results of operation. I wish now to point out the bearing of Dr. Cobb's chart on that statement. If an operation had been performed on Dr. Cobb's patients what paeans would have been sung here in praise of operation. I think we can't keep in mind too strongly the tendency to improvement without surgical interference in a certain number of apparently hopeless cases, as is shown by the history of the cases collected by Dr. Cobb and reported in his paper tonight.

DR. W. J. MIXTER: In regard to a question by Dr. Cobb as to whether surgery should come early or late, it seems to me that that question must be passed along by the surgeon to the neuropathologist to get his opinion as to whether the injury is operable if done early or whether compression of the cord can be relieved successfully at a late date. Personally, my late cases have been discouraging. The early cases have not been altogether successful either.

THREE TYPES OF SPINAL CORD INJURIES IN WARFARE. DR. HENRY VIETS.

This paper will appear in an early issue of the ARCHIVES.

DISCUSSION

DR. W. J. MIXTER: I wish to add a word to what Dr. Viets has said about the difficulties of handling such cases in war work, particularly in handling them in a casualty clearing station. In a casualty clearing station under normal conditions with a certain specified number of wounded passing through, such cases can be handled fairly well. When the press of work comes, all the fine neurologic work may break down entirely. An examination may be made in a given case in order that a decision may be reached at once, but it is impossible to pass along the information thus gained to the next man. For that reason it is practical to hold these patients as nontransportables. Hospitals for nontransportables should not be placed too close to the lines on account of shelling and since during an advance everything may be lost.

Coming naturally into this discussion is the case of a woman at present at the Massachusetts General Hospital, who was shot in the back of the neck and the abdomen. The abdominal wound was cleaned up first as was natural. The wound in the back of the neck was explored far enough to find that the second cervical vertebra was cracked but the bullet was not found. It lies in the vicinity of the second vertebra, probably just inside the canal. She has at present a paralysis of the left leg which is slight, sensory paralysis of the right leg and almost perfect sensation of the right arm. She is in

good physical condition. Her temperature and pulse are both 100. The question comes up as to what we are to do next. Are we going to try to extract the bullet or should we let it alone? The roentgen ray shows the bullet just over or just within the laminae of the second cervical vertebra.

Dr. Henry Viets: I have seen this patient of Dr. Mixter's one or twice. I think the important point is whether or not the bullet carried with it infection to the point where it now lies. If there is infection, as happened in most of the war cases, I think laminectomy is advisable. If the wound is not infected, it is a question whether to operate or not.

Dr. Stanley Cobb: In connection with the question of the noncatheterization of these patients, I think experimental evidence is of interest. I did a midthoracic cord transection on a dog. I left the dog alone hoping that in three or four days I would have an automatic bladder. I waited in vain. On the fifth day he died and necropsy showed an enormously distended bladder which filled almost the entire abdominal cavity. The bladder was so stretched that there were small hemorrhages all through it. I suppose the abnormal distention of the bladder may have paralyzed the sympathetic nerve. Other investigators who work on the spinal cord have told me that their animals die, as mine did, unless they are catheterized.

Dr. Henry Viets (in closing): Apparently the automatic bladder is of great use to the patients as months go by, but the point is that the infection is much more important than the automatic bladder. As I have tried to point out, urology should stand first and neurology second. I believe that noncatheterization is bad neurology, but if it will save the infection, it is certainly worth while.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, March 25, 1920.

G. B. Hassin, M.D., *Vice President, Presiding*

CASES WITH INVOLUNTARY MOVEMENTS FOLLOWING ENCEPHALITIS. Dr. Ralph C. Hamill.

This article is published in full in this issue.

A CASE OF LETHARGIC ENCEPHALITIS. Dr. Sigmund Krumholz.

The patient was admitted to Cook County Hospital, Feb. 13, 1920. He was 22 years of age, married, had good habits and no history of previous disease.

In the early part of October, 1919, he suddenly developed intense headache, vertigo, impairment of vision with diplopia. The headache persisted for some time and was replaced by lassitude, which gradually deepened into lethargy. In December left facial paralysis appeared, soon followed by weakness of the left lower extremity, and several weeks later by paralysis of the left arm. In January, epileptiform seizures set in, of which he had had eight by February 10. For about four weeks prior to coming to the hospital he had at times had difficulty in swallowing; choking and regurgitation of liquids. During all this time the patient was up and about until two days before admission, and he did not realize the gravity of his disease.

Examination showed a well-built, husky adult, in a stuporous state, with brief lucid intervals; the neck somewhat retracted, absence of facial expression, mouth puckered; response to questions with single words or nods of the head; with limited lateral motion of the eyes, and distinct external squint of the right eye. The pupils were unequal; they did not react to light and little in accommodation. There was spastic paralysis of the left side of the body and constant coordinate movements of the right arm and leg. The reflexes of all extremities were exaggerated but more so on the right. Babinski, Chaddock, Oppenheim and Gordon reflexes were positive on the left, negative on the right; the abdominal reflex was present. Kernig's sign was positive, Budzinsky negative; the temperature was 100.4 F., pulse 112.

Subsequent examination disclosed neuroretinitis; blood test, 4,380,000 red cells and 7,800 white cells; the differential count was normal; the Wassermann reaction negative. The urine contained a trace of albumin; no casts. The spinal fluid test showed normal pressure, clear color, positive globulin test, 15 cells; Wassermann and Lange reactions negative. Smears revealed no micro-organisms, and cultures were negative. Roentgen-ray examination of the skull was negative. Later the spinal fluid showed 8 cells.

The general condition of the patient fluctuated; the rigidity of the neck disappeared; there was transient, bilateral, external strabismus; the temperature ranged between 100.4 and 102 F.; the pulse between 100 and 144.

DIAGNOSIS: Acute and subacute meningitis as well as cerebral syphilis were ruled out chiefly because of the spinal fluid findings. Brain tumor was considered, but the presence of neuroretinitis, no choking of disks, atypical development of paralysis, and especially the constant presence of fever and rapid pulse, inclined him to a diagnosis of encephalitis.

NOTE: The patient has died and necropsy examination revealed a tumor in the region of the third ventricle, which probably explains the presence of fever.

CASE OF ENCEPHALITIS WITH UNUSUAL SYMPTOMS. DR. JULIUS GRINKER.

A woman, aged 21, married, with negative family history and unimportant personal history had a severe attack of influenza a year ago.

On the evening of Feb. 13, 1920, she complained of slight headache in the right frontal region. The next morning she awoke apparently well. Two or three hours later she vomited, became cyanotic, developed stertorous breathing with weak, irregular and rapid pulse. When put to bed, it was noted that the pupils did not respond to light and that there was convergent strabismus. The neck muscles were stiff and there was complete left hemiplegia. At this time the tendon reflexes were absent on the left and present on the right.

The next day the hemiplegia continued and the patient appeared drowsy and apathetic. She answered questions intelligently when urged to respond. To use Foster Kennedy's phrase, she was "intellectually keen, but emotionally stupid." This mental state lasted about four weeks; gradually she began to take an interest in her environment and became very much awake.

The ocular findings were transient diplopia and strabismus. Even six weeks after the onset there was occasional paresis of the left sixth nerve.

White counts were made as follows: February 14, 24,950; February 15, 15,250; February 18, 9,350; February 23, 16,550; March 8, 11,450; March 12, 8,950; March 16, 7,050.

The Wassermann test of the blood and spinal fluid was negative. The spinal fluid was under tension and somewhat blood-tinged, but contained no micro-organisms.

The maximum temperature was 101 F. Since March 2 she had had no fever. The pulse had fluctuated between 95 and 120.

On the third day of the disease the optic disks were hyperemic. One week from the onset, there was definite optic neuritis, intense within the next few days and persistent up to the present. During the past week the appearance of the disk had changed somewhat; there were to be seen whitish areas denoting scar formation and there was better definition. One could readily discern improvement from day to day.

Another unusual symptom, observed only in the polio-encephalomyelitic type of the disease and not classed under the lethargic type, was the development of muscular atrophy in the face and upper extremity. About ten days after the beginning of the disease, left-sided facial peripheral paralysis was noticed involving all the branches of the nerve. The peripheral palsy was, so to speak, engrafted on the central facial palsy.

In addition to the peripheral or nuclear palsy of the left half of the face, there were noted within a few days a certain flabbiness of the muscles of the left upper extremity, instead of the expected spasticity. Two weeks after the onset, wasting of the forearm and hand muscles on the left was pronounced and was more marked in the extensors. The biceps was somewhat wasted and felt flabby; the triceps was only slightly affected. The palm of the left hand was atrophied. The left thumb and thenar eminence were atrophic. Sensation was normal. The triceps reflex was somewhat reduced on the left, the wrist reflex completely lost.

Improvement in the paralyzed muscles was rapid. The patient could now raise the leg from the bed. The upper extremity at first improved but soon the peripheral paralysis abolished all motion. At present the arm is again improving.

The left peripheral facial palsy was still present but greatly improved and there was diplopia when the patient looked to the left. The headache, troublesome at first and almost constantly present on the right side, was still in evidence at times but the patient was happy, took an interest in everything about her and begged to be taken home.

Conclusion: The case reported partakes of the symptoms of lethargic encephalitis, hemorrhagic encephalitis, polio-encephalomyelitis, cerebrospinal syphilis and brain tumor in which hemorrhage has occurred. The Wassermann tests on blood and spinal fluid were negative and clinical signs of syphilis were absent, making that diagnosis improbable. A radiogram for evidences of brain tumor, with special emphasis on the sella and the posterior fossa, gave no clue to such diagnosis. There remained for practical consideration only epidemic polioencephalomyelitis and epidemic encephalitis of the lethargic variety. Though lethargy was a pronounced symptom in this case, we were not dealing with an encephalitis only, but also with a poliomyelitis; in other words, a lethargic polio-encephalomyelitis.

A CASE OF THIRD VENTRICLE TUMOR MISTAKEN FOR LETHARGIC ENCEPHALITIS. Dr. Peter Bassoe.

A woman, 53 years old, was admitted to the Presbyterian Hospital on Jan. 14, 1920, Three or four months previously she became irritable, abrupt and seclusive, and lacking in self-confidence. On December 31 she failed to prepare supper for her husband, insisting that the meal had already been eaten.

The next day she complained of frontal headache; slept poorly for a few nights and rapidly became more confused. On January 7 she became bedridden and thereafter did not recognize people. Her conversation was always incoherent. She did not complain of headache except on the date mentioned. When examined on January 15 she was semistuporous and muttered incoherent words. She was rigid and constantly clenched the fists. The pupils and disks were normal. The facial expression was rather tense. No paralysis could be discovered. The knee jerks were weak; the right ankle jerk was present; the left absent. Abdominal and arm reflexes were not obtained.

Hemoglobin was 75 per cent., erythrocytes 4,600,000; leukocytes 13,400.

Spinal Fluid: The Wassermann test was negative; the Ross-Jones globulin test positive; cell count 98, mostly lymphocytes; Lange gold test: 0112332100.

Gradually she became more stuporous and more rigid and perspired freely. After a few days a Babinski sign was obtained on the left side and later on both sides. January 19 it was noted that the right eye was more widely opened than the left. The condition of the tendon reflexes varied; at times none was obtained. February 10 moderate left ptosis was recorded. Although very stuporous she could be made to eat quite well. Continued fever was present from the date of admittance, the temperature varying from 100 to 101 F. during the first week; after January 21 it ranged from 101 to 103 F. The pulse rate was variable, becoming rapid toward the end. A second lumbar puncture on February 5 yielded a clear fluid with a cell count of 5, weakly positive globulin test, and a weaker Lange reaction (0012221100). The patient died on February 20. The clinical diagnosis was lethargic encephalitis, and no other condition was seriously considered. It is regrettable that the eyegrounds were examined on only one occasion.

Necropsy Findings: The brain weighed 1,507 gm. after three weeks of hardening in formaldehyd. The pial veins were moderately distended, the convolutions broad and flattened, giving an external appearance of edema. There was some milky thickening along the superior longitudinal fissure.

On section the white matter in the frontal region showed decided hyperemia. Section across the anterior end of the lateral ventricles showed the left frontal horn to be markedly distended and the right one moderately so. A broad, grayish-red tumor mass formed a barrier between the two ventricles, being attached to the corpus callosum above and the orbital convolutions and the caudate nuclei below. It merged imperceptibly into the brain substance below, but was sharply defined from the corpus callosum. At this level the tumor measured 3.5 cm. from side to side and 2 cm. from above downward. A small cyst 2 mm. wide was seen in the tumor tissue.

A section through the infundibulum showed that the tumor filled the third ventricle and on the left side an oblong cyst 5 by 9 mm. projected into the left lateral ventricle; while a small sessile nodule projected into the right ventricle. The left foramen of Monroe was open, while the right one was slit-like and compressed by the tumor. The base of the tumor on the right side merged with the innermost portion of the basal ganglions and internal capsule. The tumor ended 1 cm. behind this level, the posterior part of the third ventricle being free. The aqueduct was considerably dilated. The lateral ventricles at this level were only slightly dilated, while farther back the occipital horns were much dilated. The most posterior part of the tumor contained a cyst which measured 12 by 8 mm.

DISCUSSION

Dr. G. B. Hassin thought the pathologic changes in a typical case of lethargic encephalitis were present in the cortex as well as in every other part of the central nervous system, affecting both parenchymatous and interstitial elements. The most striking parenchymatous changes show as neuronophagia, which is considered by some observers a rare or accidental phenomenon in encephalitis. In six cases studied by him, neurophagia was a constant finding. It was also present in Bassoe's atypical case of encephalitis and will be found in any other case, especially in the deeper cortical layers adjacent to the white brain substance, provided a sufficient amount of slides are studied. In an acute disease like lethargic encephalitis, lasting days or weeks, one cannot expect such widespread changes as in dementia paralytica or African sleeping sickness, which are chronic diseases of years' duration. Even in these types of encephalitis, many ganglion cells may be found in a practically normal condition. Absence of parenchymatous changes is rather inconsistent with the diagnosis of encephalitis. Their unusual localization or severity may be responsible for the unusual symptoms in cases like those of Bassoe, Hamill and possibly Grinker. Unfortunately, pathologic studies of such abnormal localizations are lacking, Dr. Bassoe's case being so far the only one more or less carefully studied. Pathologic studies are absolutely essential for establishing the true nature of many atypical cases reported and demonstrated as lethargic encephalitis, some that are decidedly another condition, for instance, a tumor.

Dr. Julius Grinker stated that a patient with lethargic encephalitis under his care at present had often made him think of brain tumor that might give similar symptoms, and if the patient went to exitus he would not be surprised to find the same things that Dr. Bassoe had described. At present the patient was making a splendid recovery. He thought the diagnosis should be made with great caution, but as a rule one need not wait for postmortem findings to diagnose lethargic encephalitis.

Dr. A. S. Hershfield asked Dr. Bassoe what the eye findings were in the case of tumor.

Dr. Bassoe replied that the eyegrounds were normal when the patient was admitted to the hospital, but they were not examined again.

Dr. Grinker asked whether any one had ever seen optic neuritis in poliomyelitis.

Dr. Hamill replied that he saw a case in a child 1½ years olds, who had typical poliomyelitis with choked disk. Lumbar puncture was done and the necropsy showed marked polio-encephalitis.

Dr. Grinker said that about three years ago he sent a woman of 30 into the Cook County Hospital in a state of infectious delirium. Examination revealed typical choked disk and slight atrophy of the hand muscles and marked foot drop on one side. The patient was blind. At that time Dr. Nuzum was making his first experiments with the serum treatment of poliomyelitis and he administered two doses to this patient. She made a perfect recovery and could see again. The case was a typical polio-encephalomyelitis with optic neuritis and it was his experience with that case which enabled him to reconcile the optic nerve findings with the diagnosis of lethargic polioencephalomyelitis in the case just reported.

Dr. Hugh T. Patrick thought the most confusing thing about lethargic encephalitis was the multiplicity of types. Some of the cases began with intense headache, other patients did not have headache. Some, as Dr. Grinker said, presented quite a typical picture of toxic delirium, in others the condition was strongly indicative of meningitis, with marked retraction of the head, stiffness of the neck and a Kernig's sign, with rather clear sensorium. Another peculiar mental state was one of apparent lucidity with severely impaired circulation. He had seen one young man with marked retraction of the head, a distinct Kernig's sign and feeble reflexes, fever not high, who seemed quite clear mentally. He looked alert and answered questions as to how he felt, quickly and correctly, presence of pain, etc., but when asked where the pain was located he did not know. He did not know he was in a hospital, or what city he was in and could give no account at all of the last two weeks.

He had seen a similar case in an older man. His family said he was all right mentally, but questioning revealed that he was disoriented and badly confused about familiar things. This case was quite suggestive of rapidly progressive vascular trouble, especially as he had had several peculiar attacks of transient dizziness and confusion.

Then there was the type with rigidity and facies of the Parkinson type; these patients were generally quite rational and answered questions intelligently. At the time he saw one patient of this type an attorney was there to draw the will and it was obvious that the man was competent to make a will. He answered questions concerning finances and what he wished done with entire lucidity, and yet when he was let alone, one would say that he was probably semiconscious if not unconscious; but that was in appearance only.

Some of the cases looked much like cerebral or cerebrospinal syphilis, and some apparently had a marked functional element. Dr. Patrick had recently had under observation a woman who acted like an old hypochondriac or psychasthenic; she worried constantly about herself and repeatedly asked him whether he was sure she was not going to die. This patient had persistent tachycardia, with no apparent cause. Some patients complain of numbness, tingling and dryness of the mouth, others develop peculiar contractures and still others odd tremors or movements reminding one of athetosis or chorea.

Dr. Patrick asked Dr. Bassoe whether he had formulated anything on which he could base a prognosis, and confessed that personally he could do nothing but guess at the outcome. He cited two contrasting cases: A young man was decidedly disoriented, with marked Kernig's sign and strongly retracted head. He was apparently very ill and made a rapid recovery. He had been seen only two and a half months ago; since that time he had recovered, married, gone on a wedding trip, returned and was at work. At about the same time he saw a young woman in about the same condition, except that she had severe headache and was slightly delirious, though she could be roused and answered questions rationally. She died four or five days later. When seen her condition did not appear to be much more serious than that of the young man.

Then there were the patients who passed through the acute stage and went along with some disability. What was the ultimate outcome in these cases to be? Would they be left with some physical or mental impairment, or both?

Dr. Patrick also asked whether Dr. Bassoe had found any treatment that was of value.

Dr. Bassoe said he could not answer Dr. Patrick's question in regard to prognosis and treatment.

CHICAGO NEUROLOGICAL SOCIETY AND CHICAGO PATHOLOGICAL SOCIETY

Joint Meeting, April 12, 1920

LEWIS J. POLLOCK, M.D., *President of the Neurological Society, in the Chair*

THE MORPHOLOGY OF THE SYNAPSE IN VERTEBRATES.

GEORGE W. BARTELMEZ, Department of Anatomy, University of Chicago.

In no field of investigation has the personal factor played a more prominent part than in the study of the ultimate structure of the central nervous system, especially in the matter of the synapse. The chief reasons for this are the complexity of the tissue, the minute proportions of the structures involved with the attendant difficulties of interpreting the microscopic picture and also the prominence which technical procedures must play in the study. Since the neuron theory accepted the Cajal doctrine of the synapse by contact it has been most successfully attacked from this aspect. If one be inclined to question whether the Golgi and Cajal methods give complete pictures of the strictly nervous elements of the tissue, then the bulk of evidence at the present time favors protoplasmic continuity at the synapse. This is significant since the findings favoring continuity are positive as contrasted with the negative findings for contact. On the other hand, our present knowledge of the structure of protoplasm and of the effects of current histologic methods on it are beginning to imbue us with a hopeful skepticism concerning the reliability of most cytological preparations, particularly of the central nervous system. We need more perfect methods and there can be little doubt that, when developed, they will discredit much generally accepted work.

No other tissue approaches the central nervous in complexity, so it is worth while to enumerate the number of histologic elements which enter into the composition of the gray matter. Five groups of structures are recognized:

(*a*) The undoubtedly nervous elements: the perikarya, their dendrites, axons with collaterals and the ramifications of afferent axons.

(*b*) The neuroglial elements: the protoplasmic glia syncytium which is probably the Golgi net of Bethe, the neuroglia fibers and in most preparations the products of their disintegration.

(*c*) Myelin sheaths or the products of their partial solution such as neurokeratin nets.

(*d*) The fluid substratum or ground substance which certainly contains coagulable proteins.

(*e*) Blood vessels with their accompanying collagenic tissue.

All of these must be taken into account and besides there are the distortions following the solution of various elements and the shrinkage due to dehydration and embedding which add to the difficulty of interpreting the microscopic picture. It is essential that very thin sections be used if reliable observations are to be made and even then it is difficult to understand oblique sections.

Nowhere has the personal factor so insidiously entered into the problem as in the technical procedures. Since no one has thus far been able to study a living uninjured nerve cell or the effects of various fixing fluids on it, the criteria of a well preserved preparation have remained largely subjective. With the development of special methods for the various constituents of nervous tis-

sue the tendency has grown to place implicit faith in the microscopic preparation. The study of many methods has convinced me that in most types of synapses the appearance of contact or continuity can be produced by using appropriate fixatives or by sufficiently hydrating the coagulated protoplasm with long washing, treatment with alkaline solutions, etc. Thus there can be no doubt that the descriptions of so careful an observer as Hans Held are perfectly objective, but I believe that his methods have produced fundamental changes in the relations of the nervous elements in the synapse. All who have worked largely with material fixed in formol and especially with the

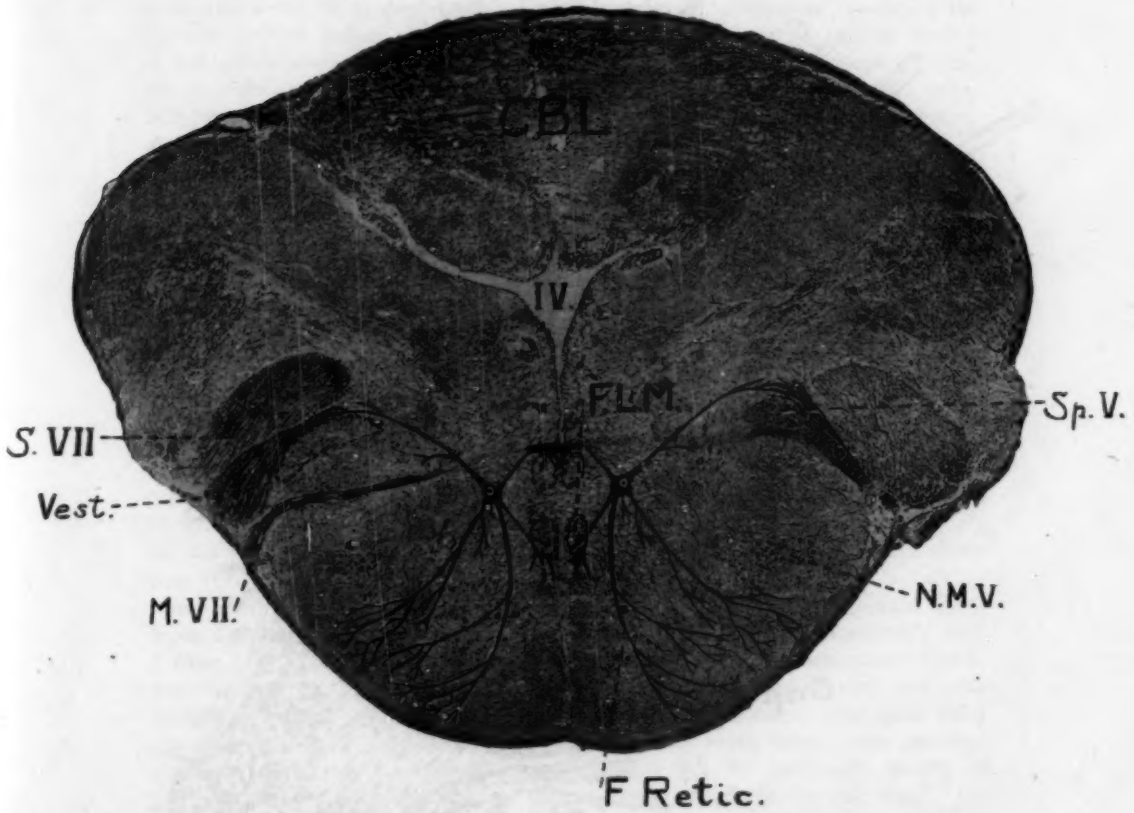


Fig. 1.—A photomicrograph of a section taken through the brain of a catfish at the level of the two Mauthner's cells. These cells have been reconstructed from the neighboring sections and projected into a single section. The ventral dendrites spread through the whole formatio reticularis. The lateral dendrite extends as far as the spinal fifth tract. It is surrounded by the fibers of the vestibular nerve, certain of which terminate on it in large club endings. The axons of the Mauthner's cells cross immediately and extend throughout the spinal cord in the fasiculus longitudinalis medialis. *CBL*, cerebellum; *F.L.M.*, fasiculus longitudinalis medialis; *F.Retic.*, formatio reticularis; *IV*, fourth ventricle; *M.VII*, motor root of N. facialis; *N.M.V.*, motor nucleus of N. trigeminus; *Sp.V*, spinal fifth tract; *S.VII*, sensory root of N. facialis; *Vest.*, vestibular root fibers.

Bielschowsky technic have supported the continuity theory, a fact which is to be associated with the swelling effect of the formol solution on nerve fibers even when it has been "neutralized" with magnesium carbonate. The only material in which nothing is stained but indubitable nervous structures is a successful Cajal preparation. Of course in the case of most synapses there is no answer to the criticism that the method does not stain *all* the nervous elements; but in at least one case there is an answer, as will appear shortly.

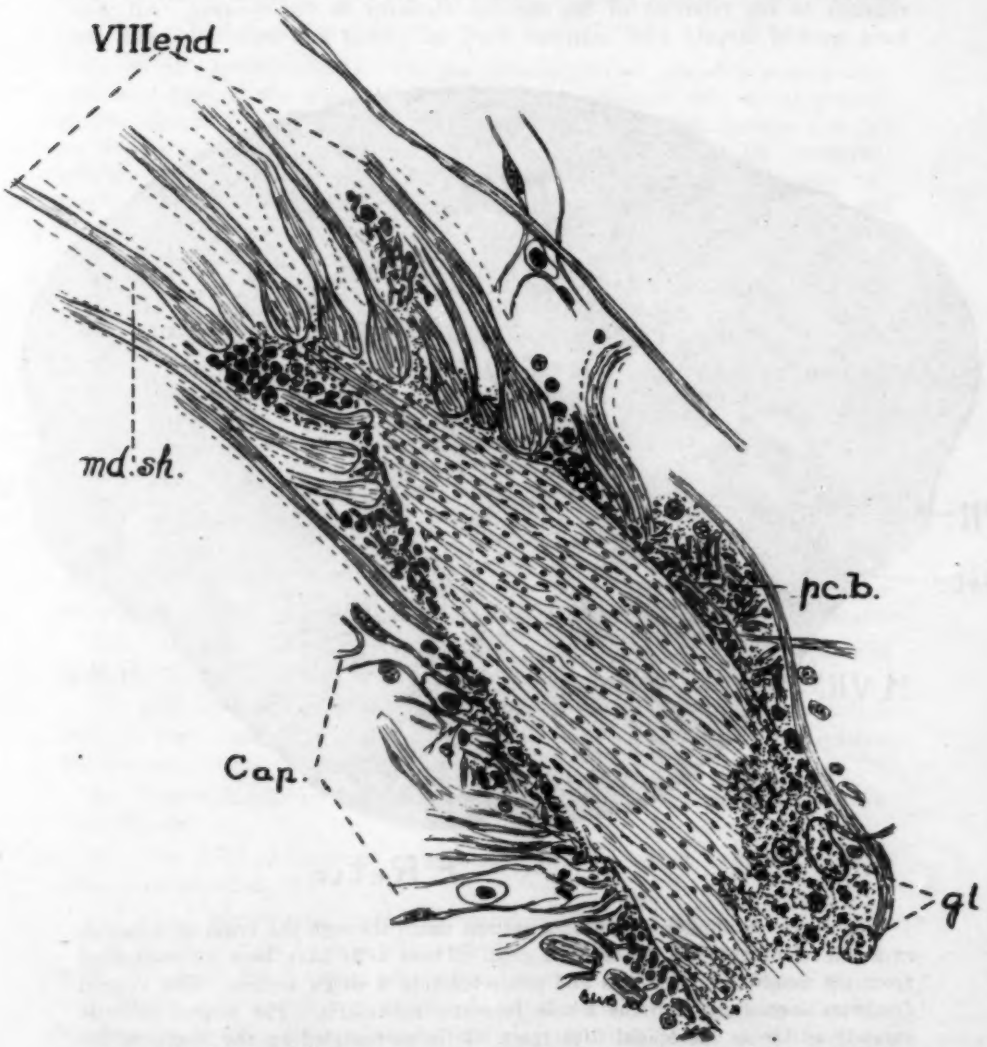


Fig. 2.—A portion of the lateral dendrite of the left Mauthner's cell of a catfish with the club endings of the thick vestibular root fibers ending on it; × 525. In the lower right hand corner of the figure the pericellular basket is shown in surface view where it spreads over the obliquely cut dendrite. Two glia nuclei show here at the edge of the dendrite. *Cap.*, capillaries; *gl.*, neuroglia nuclei; *md.sh.*, medullary sheath of a root fiber; *pc.b.*, pericellular basket; *VIII, end.*, vestibular root fibers with club endings.

Most other preparations show additional elements, thus complicating the picture and these are preferred by the advocates of continuity. Metallic impregnation methods come nearest to giving a specific stain for nervous elements but even here the specificity is only relative. Slight changes in the method may give very different pictures and when the glial elements are visible it is usually impossible to distinguish them from nervous elements by color alone; no matter how good the preparation, it is never possible so to differentiate them throughout an entire cross section of the brain. In any case it is essential that the silver methods be checked by other methods which have been shown to produce minimal change in form relations when used on tissues which can be studied fresh. Unfortunately the ubiquitous lipoids of the nervous system reduce many of the reliable fixing agents so rapidly that it is impossible to obtain adequate fixation even in small pieces of tissue. Unless the lipoids are at least partly preserved, the distortions due to their solution are so great as to invalidate any conclusions concerning the minute structure of most synapses.

Up to the present time only one synapse has been found in which the elements are large enough to give unambiguous microscopic pictures in all types of preparations. In 1915 I called attention to certain large bulbous endings of the vestibular nerve on the lateral dendrite of Mauthner's cell in the catfish, *Ameiurus melas* (*J. Comp. Neurol.* **25**:87, 1915). Here giant vestibular fibers terminate in club endings, the largest of which are 7 microns, the average being 4.6 microns in diameter as measured in sections. The endings lie in contact with a sword-shaped dendrite of a large cell, the axon of which extends throughout the spinal cord and makes direct connections with all the motor centers concerned in swimming. It readily shows the effects of adverse conditions as do other highly specialized cells in the nervous system. Marui (The Finer Structure of the Synapse of the Mauthner Cell, *J. Comp. Neurol.* **30**:127, 1919; ibid. **30**:253, 1919) has studied Mauthner's cell and some of its synapses under both normal and experimental conditions, but unfortunately he did not find these endings on the lateral dendrite which would have given him really clear evidence. This failure is probably due largely to a lack of familiarity with the teleost brain. Since he came to the conclusion that fibers from the dense pericellular feltwork enter into the body of Mauthner's cell, I have gone over the field again and come to the conclusions stated above; namely, that the technic plays a decisive rôle in the study of such synapses as Marui worked with. In thick sections (8 to 10 microns) of formol fixed material, especially in Bielschowsky and Levaditi preparations, one can find pictures which may be interpreted as evidence for the continuity theory of the synapse, but only in the very delicate synapses about the cell body. On the other hand, in every preparation I have studied, and this includes formol material, the histologic picture of the club endings on the lateral dendrite can be interpreted in only one way: viz., the vestibular endings in every case lie close to the lateral dendrite but never show any indication of fusion with it. This leads me to view with the utmost skepticism the reality of the conditions which have been observed by Held, Bielschowsky, Bethe, Marui and others in histologic preparations where there was continuity between different neurons (Heidenhain: Plasma und Zelle in Bardeleben, Handbuch der Anatomie des Menschen, **8**). That is to say, I believe that Held's pericellular nervous terminal net, the fusion of the protoplasm of "endfeet" with that of perikarya, the entrance of pericellular fibrils into the substance of nerve cells and similar conditions are postvital fixation or maceration artefacts. Experiments are in progress to test these conclusions in greater detail.

Marui (1919) has recently published the first study on the synapse under experimental conditions. In the much more complex synapses of Mauthner's cell which he studied, no changes in the purely nervous structures were found. His findings in the activities of the glia cells under experimental conditions in exhausted animals are of prime importance, but it remains to be seen whether these were due merely to the harsh method of "fatiguing" or to serious injury of nutritional origin.

DISCUSSION

DR. C. JUDSON HERRICK said Dr. Bartelmez was frank in expressing himself as a neuronist, with the preconceptions which this implies. He thought the majority of neurologists had been, or still are, neuronists. The facts in the case were clear in this particular instance. The histologic pictures, which he had personally examined, were as unambiguous as the histologic pictures one was in the habit of getting in studies on other tissues.

One thing must not be forgotten: Suppose we have a membrane which bounds the protoplasm of the end of the axon as it comes in from the vestibular nerve, and a second membrane which bounds the cell body to which this ending is applied, the nature of these membranes must not be neglected; they are not inert substances, but living tissues. It is still an open question whether to speak of protoplasmic continuity or not. The preparations appeared to show that there were two kinds of living substance intervening between the protoplasm of the first neuron and that of the second. That there was something there which was of tremendous physiologic importance, every one believed, and if we have a contrast between the protoplasm of the first neuron and that of the second and a further contrast between the two protoplasms that constituted the two membranes, there was some ground for the doctrine of discontinuity. Nevertheless, they were all living substances and so it was largely a question of definition whether to speak of protoplasmic continuity or not.

THE RELATION OF THE CEREBRAL HEMISPHERES TO ARTERIAL BLOOD PRESSURE AND BODY TEMPERATURE REGULATION.

F. T. ROGERS.

This article will appear in an early issue of the ARCHIVES.

DISCUSSION

DR. S. A. MATTHEWS said that Isenschmid and Kiehl (*Arch. f. exper. Path. u. Pharmakol.* **70**:109, 1912) have published experimental results indicating that the caudate nuclei and the anterior part of the optic thalami may be recovered from rabbits with only a temporary change in the heat regulatory mechanism. When, however, the posterior and ventral portions of optic thalami were removed the animals became poikilothermal. Also Citron and Leschke (*Ztschr. f. exper. Path. u. Therap.* **26**:161, 1913) assert that destruction of the median portion of the between brain on the boundary of the corpus quadrigeminum and the optic thalami anterius makes rabbits poikilothermal.

In dogs it is quite easy to gain access to the third ventricle by drilling a hole about 5 mm. in diameter through the sphenoid bone just back of the optic chiasm. Of course, this will encroach somewhat on the pituitary body, but through such an opening lesions can be made in the basal ganglions.

In one dog in which he made a lesion in the anterior part of the optic thalami and which involved the principle, accessory and dorsal nuclei of the tuber cinereum, and after which the animal lived for three weeks, the temperature gradually fell and during the last two days of life was as low as 22 C. The animal gradually developed a lethargic condition.

When the animal was placed in an incubator at from 31 to 32 C. the temperature would go up to normal, and the reflexes would approach a normal state.

Evidently, from Dr. Rogers' results on birds and the results of others on mammals, the thalami contain the seat of the heat regulation. Just how they bring this about, whether by increased heat dissipation or by diminished oxidation has yet to be shown.

Dr. I. Leon Meyers asked whether any examinations of the temperature or arterial pressure had been made in dogs after producing in them the state of decerebrate rigidity.

Dr. Lewis J. Pollock asked what was done in these operations relative to closing the cranium so as to keep intact the intracranial pressure as this would probably have considerable to do with the general blood pressure.

Dr. F. T. Rogers (closing) said that the three questions are all significant. In reference to Dr. Meyers' question, this point has been tested out in the dog and it is known that the loss of the cerebral hemispheres leads to a depression of the blood pressure. In the dog there are certain complications however, due to anesthesia and to changes in the muscle activities involved in decerebrate rigidity.

Dr. Matthews has struck the keynote of the problem in referring to calorimetric controls in this work. It is evident that in the temperature regulation one must consider both heat production and heat loss. The heat loss in the bird seems to be principally through the skin and secondarily through the lungs by expiration. As to heat production, he was inclined to think, on the basis of evidence not yet conclusive, that there is deficient heat production resulting from diminished muscle activity.

As to Dr. Pollock's remark, his is the same question that has been raised by others in the last few years. In an article recently published by L. M. Moore, it is stated that a rise in body temperature does not follow puncture of the corpus striatum unless it is associated with increased intracranial pressure. In this report cases are not included in which there is clear evidence of increased intracranial pressure.

Furthermore, it may be added that it is an open question as to how much of the effects of hypophosectomy or irritation in the hypophoseal regions is due to secondary disturbances of the adjacent brain stem.

STUDIES ON THE SYMPATHETIC NERVOUS SYSTEM. S. Walter Ranson.

It is probable that the sympathetic nervous system is constructed along somewhat simpler lines than those indicated by the description found in most textbooks of histology. It is well established that visceral efferent fibers run through the ventral roots and white rami to the sympathetic trunk, within whose ganglions the majority of the fibers end. Some are continued through the splanchnic nerves to the ganglions of the celiac plexus. These fibers end in synaptic relation with neurons of a second order, often designated as sympathetic neurons, whose axons run to end on involuntary muscle or glandular

tissue. Thus two visceral efferent neurons are concerned in transmitting an impulse from the cerebrospinal nervous system to smooth muscle or gland, and these are often designated as preganglionic and postganglionic with reference to the ganglion which contains the synapse between them.

There are probably no afferent neurons in the ganglions of the sympathetic trunk nor in the collateral ganglions, and no afferent fibers running from these ganglions to end in the spinal ganglions. Dogiel's work, published in 1896, in which he distinguishes both sensory and motor neurons, has not been supported by more recent investigations. Langley has shown, and we have been able to confirm his observations on this point, that after section of a spinal nerve distal to the spinal ganglions all or nearly all of the fibers in the corresponding white ramus degenerate, which would not be the case if any considerable number of afferent fibers of sympathetic origin were directed through the rami into the spinal ganglions. The afferent fibers of the sympathetic trunk and splanchnic nerves are probably all derived from cells in the spinal ganglions.

Sections of the superior cervical ganglion of man, stained by the pyridin silver technic, display a wealth of dendritic branches arising from the multipolar ganglion cells. These are of two kinds: namely, subcapsular dendrites and extracapsular dendrites. The latter pierce the membranous sheath or capsule, which surrounds the cell, and run long distances in the ganglions, branching repeatedly. The subcapsular dendrites branch and intertwine beneath the capsule, causing it to bulge locally and forming what may be called a glomerulus. When the localized subcapsular mass of dendritic ramifications is formed from the dendrites of one cell, it may be called a simple glomerulus. If formed by dendrites from two or more cells it may be designated as a bicellular, tricellular or multicellular glomerulus.

The extra capsular dendrites interlace with untold numbers of fine branching axons, forming an intercellular plexus which occupies the interspaces among the ganglion cells. We are interested chiefly in the axonic ramifications which help to constitute this plexus. Huber and Dogiel asserted that the majority of these fibers were the axons of sympathetic cells in the same or nearby ganglions; and their observations on the composition of this plexus constitutes the only histologic evidence worth mentioning for the existence of association neurons in the sympathetic system. Such neurons, if present, would serve to unite one ganglion with another or the several parts of one ganglion with each other. The physiologic evidence, so well presented by Langley, is all against the existence of such association neurons. The important question then is: What is the source of the fine axons in this plexus?

In a number of dogs we cut the cervical sympathetic trunk, thus severing all of the preganglionic fibers which run to the superior cervical ganglion. After time was allowed for degeneration, pyridin silver preparations were made of the superior cervical ganglion. These showed a complete absence of the fine branching axons of the intercellular plexus. These experiments furnish strong evidence that no synapses are present in this ganglion except those between preganglionic and postganglionic neurons.

The sympathetic trunk of the cat consists of more or less segmentally arranged ganglions bound together by fibers from the white rami. Preganglionic fibers from the upper nine white rami run upward in the trunk to the stellate and superior cervical ganglion. Similar fibers from the white rami below the fourth run downward in the trunk and many of these run through the splanchnic nerves to the celiac plexus. Others terminate in the ganglions of the thoracic, lumbar and sacral portions of the trunk.

The visceral afferent fibers of the sympathetic trunk and splanchnic nerves take origin from the cells of the spinal ganglions. They include large, small and medium-sized myelinated fibers and many which are unmyelinated. These were studied in white rami from which all the preganglionic fibers had disappeared as a result of section of the corresponding spinal nerves proximal to the spinal ganglions. Such partially degenerated rami containing only sensory fibers could be followed into the trunk by a study of serial sections. In the trunk each such ramus forms a longitudinal fascicle which maintains its identity for long distances, and the fascicles from the lower thoracic white rami can be traced into the splanchnic nerves.

The splanchnic nerves of the cat usually contain no appreciable number of postganglionic (i. e., sympathetic) fibers. These nerves are composed of preganglionic visceral efferent fibers from the spinal cord and visceral afferent fibers from the spinal ganglions, both of which reach the sympathetic trunk by way of the white rami and merely run through that trunk to enter the splanchnic nerves. In two of ten greater splanchnic nerves examined there were also present small bundles of postganglionic fibers. In one of these instances it was obvious that these fibers arose from an extra ganglion located on the sympathetic trunk between the first and second lumbar sympathetic ganglions.

These observations have been reported in greater detail in volume 29 of the *Journal of Comparative Neurology*.

DISCUSSION

Dr. C. Judson Herrick asked whether Dr. Ranson had extended his observations to the peripheral ganglions, and if he could give any suggestions as to whether they would apply equally to the ganglionic complexes of the abdominal viscera, etc.

Dr. Ranson (closing) thought Dr. Herrick's question was pertinent. He was sorry to say that he had not been able to get a satisfactory preparation of the peripheral ganglions. The technical difficulties involved in the study of the peripheral ganglions are much greater than those encountered in the study of other parts of the sympathetic system. He had tried to be careful to limit his remarks to the sympathetic chain, splanchnic nerves, and the prevertebral ganglions. In regard to these, the physiologic and histologic evidence is in full accord; but in the case of the peripheral ganglions, the physiologic evidence seems to point to something which we do not have in the ganglionic chain, a mechanism for visceral reflexes that is independent of the cerebrospinal axis.

THE NEW YORK NEUROLOGICAL SOCIETY

The Three Hundred and Eightieth Regular Meeting, April 6, 1920

Walter Timme, M.D., *President, in the Chair*

PRESENTATION OF CASES OF FAMILIAL DYSTONIA MUSCULORUM OF OPPENHEIM. Presented by Dr. Isadore Abrahamson.

Dr. Abrahamson presented three patients in one family, illustrating different stages of the disease. They were of the pure idiopathic variety, noteworthy especially, first, for their definite familial character; second, for their resemblance in the deviations from type that are to be encountered in all familial dis-

eases; third, for a distinct involvement of speech, which Oppenheim denied in his cases; fourth, for the involvement of the musculature of the neck not common in these cases; fifth, for the varying mental attitude in the three patients, and sixth, for an unusual propulsive phenomenon which had not previously been noted in cases of this sort.

The first patient, a young Russian woman of 20, with unimportant family and personal history, at the age of 12 experienced difficulty in writing and became clumsy in her gait. The muscles of her legs would stiffen, the stiffening gradually spread to other muscles and was particularly increased by effort or emotion. While trying to lie quietly her muscles stiffened and caused her extreme annoyance. On attempting to grasp an object, a coarse tremor appeared. Her symptoms became aggravated during the last three months before presentation so that she could neither sit nor walk. During sleep all symptoms disappeared. The muscles were hypotonic. There was no paralysis. An abnormal wrinkling of the forehead and general anxiety were deviations from the usual Oppenheim manifestations. Speech was dysarthric, of bulbar type. The legs were paralytic, usually kept crossed, the typical inward rotation of the thigh was clearly apparent, and the feet were turned down. The Adam's apple was prominent, and she showed torticollis. Dystonia, tortipelvis, lordosis, clonic movements of the left hand with tonic movements in legs, and a rhythmic tremor characterized the disease in this patient.

In the second sister, aged 16, the disease had an insidious onset. She first noticed difficulty in writing, her muscles began to stiffen involuntarily, the left arm was drawn up in flexor spasm, the hand turned outward. She complained that the hip joint on the right would not stay in place, and a drawing feeling above the knee caused her to incline forward, producing a type of propulsion that is very interesting. Her body was thus bent forward and also laterally twisted around the vertical axis of the spine. There was inward rotation of the thigh. The gait was bounding. Her condition was much improved when feeling well and rested.

The third member of the family, a boy of 15, showed the disease in its early stages. His first difficulty was also with writing. He had the inward rotation of the thigh, the toes pointed down, the heel was carried high. This condition had slowly been getting worse for the last two years. In walking he swished his foot, and there was a slight forward bend. He ran well but had difficulty in stopping. He could make some hand movements such as threading a needle and playing marbles. A fine tremor was noted. Scoliosis and tortipelvis were present.

The three patients showed interesting differences in emotional states. The first was extremely anxious, emotional and worried about her condition. The second was optimistic and wanted to get well. The boy was apathetic, had no interest in his condition, and in general showed the mental state usually found in such cases, which sometimes causes the patients to be classed as hysterias.

Dr. Smith Ely Jelliffe thought it was unique to have three members of a family showing the beginning, middle and developed phases as had been demonstrated. Dr. Abrahamson had mentioned Ziehen's familial group, which Dr. Jelliffe had had the good fortune to study in Berlin. There were three in that group also, and Schwalbe's monograph had presented the features, but in Ziehen's group the disorder had progressed to a more or less uniform pattern and no developmental study was possible such as the present presentation offered.

Dr. Jelliffe was disposed to emphasize the varying clinical trends of a large group of striatum syndromes of which these cases were but one of the striking types. It had become increasingly evident that dystonia musculorum, Vogt's double athetosis, Westphal's pseudosclerosis, Wilson's lenticular degeneration, Huntington's chorea, tuberous sclerotic idiocy, and even paralysis agitans were to be regarded as but variants in this larger picture of striatum syndromes.

It was recalled that one of Ziehen's patients had come to necropsy and negative findings had been reported, but more recently one of Flatau and Sterling's cases had been examined postmortem, and Thomalla,[1] Schneider[2] and von Economo[3] had respectively studied the striatum pathology and the liver, for the case of Flatau and Sterling had afforded a combination of the Ziehen-Oppenheim group of dystonia musculorum types and Wilson's lenticular degeneration types, since the clinical picture was characteristic of the former trend, while the hypertrophic sclerosis of the liver was of the Wilson lenticular degeneration type. The pathologic picture of the striatum was one of abiotrophic atrophy of the cells of the putamen.

Vogt[4] and Bielschowsky[5] quite recently attempted a synthesis of the various striatum syndromes outlining the pathologic trends and enlarging the neurologic pictures to be comprised in the group. Thus the Vogts distinguish (1) the *état marbé*, which they originally described as double athetosis and in which group many instances of Little's disease are to be included; (2) the *état fibreux*, which progresses often as a bilateral progressive chorea without psychic symptoms, also shown in Huntington's chorea; (3) total necrosis of the striatum, chiefly confined to the putamen, and at times reaching to other parts of the globus pallidus. To this group the dystonia case of Thomalla belongs and Wilson's lenticular degeneration. (4) Acute infectious striatum syndromes; many of these have been observed in our recent lethargic encephalitis epidemic as paralysis-agitans-like cases, athetotic cases, pseudobulbar palsies, involuntary laughing and crying cases, etc. It would lead too far to take up Bielschowsky's grouping of the various syndromes, and Dr. Jelliffe referred to these recent studies.

One point of speculative interest Dr. Jelliffe said he would like to raise, namely, the opportunity here offered to study the syndromes now under discussion and their relationships and that of the striatum to the vegetative control of muscle metabolism through the liver. He spoke of correspondence with Wilson relative to the evidence afforded by lenticular degeneration and liver metabolism. Whereas Wilson had argued for a toxin from perverted liver function as causing the striatum changes, Jelliffe would emphasize the neural integration disturbance of striatum interruption of vegetative liver pathways. The case of Thomalla, in which the liver was carefully studied by Schneider, offered several interesting points bearing on the possibilities of an analysis of the vegetative integration mechanism which was essential for the liver function, interference with which caused the vagotonic gliosis, and this put the entire muscular metabolic functions to disadvantage, in that the integration of the involuntary part of the muscle structure, the sarcoplastic substance and the

1. Thomalla: Ztschr. f. d. ges. Neurol. u. Psychiat. **41**: 1918.
2. Schneider: Ztschr. f. d. ges. Neurol. u. Psychiat.
3. Von Economo: Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.
4. Vogt: Jour. f. Psychol. u. Neurol. **24**:1, 1918.
5. Bielschowsky: Idem. **24**:20 and 28, 1918.

sensori motor anisotropic disk parts were seriously impaired. Here was an important field in which future studies could be prosecuted and the vegetative arc components, their spinal pathways and higher integrative synaptic connections laid bare.

Dr. Tom A. Willaims of Washington (by invitation) spoke of the physical components of muscle and the mechanism of muscular tonus. The chemistry of muscle reaction and phases of metabolism have been dealt with by Bjerron.

These cases, Dr. Williams felt, did not show true propulsion, since they were able to arrest themselves and turn without difficulty. The attitude assumed by the second patient in walking caused a sort of propulsion. The boy, however, could go upstairs and walk and run. The peculiarity of gait is a hypotonus rather than a true propulsion. Wherever there was voluntary increased tonus the propulsive phenomena would seem to disappear. In other words, the phenomena observed might be termed pseudopropulsion due to dystonia, overcome by volition.

Dr. Abrahamson, in closing, explained that clinical propulsion, such as was seen in Parkinson's diseases and in certain cerebellar affections, was dependent on the abnormal attitude of the patients or to a faulty gait.

True propulsion or forced movements resulting in propulsion were to be found in lesions affecting the pathways subserving static equilibrium, i. e., the semicircular canals — vestibular nerves, Deiter's, Bechterew nuclei, postlongitudinal bundles or interstitial nucleus of the thalamus. Experimental work on this pathway, in particular the work of Muskens on the posterior longitudinal bundle, proved this. In the cases of dystonia shown the patients exhibited the clinical type of propulsion and not the forced movement type.

AN ANALYSIS OF THE CASES ADMITTED TO THE NEUROPSYCHIATRIC SERVICES OF THE U. S. ARMY GENERAL HOSPITAL NO. 1 (COLUMBIA WAR HOSPITAL, NEW YORK CITY).

Dr. Sylvester R. Leahy.

This article will be published in full in the Archives.

ACUTE DESCENDING RADICULITIS — A SPINAL TYPE OF EPIDEMIC ENCEPHALITIS.

Dr. Irving H. Pardee.

This article is published in full in this issue.

A STUDY OF PUBERTAS PRAECOX AND THE MENTALITY OF THE PATIENTS.

Dr. J. H. Leiner.

Dr. Leiner reviewed the historical references to pubertas praecox. In this syndrome it would seem that the child passed through several stages of life in utero. The endocrines seem to be a primary factor in the causation of the condition. Secondary factors are climate, race and heredity. As is well known, menstruation appears normally at a somewhat earlier age among women in southern countries, while the inhabitants of the north normally do not begin to menstruate until after the age of 14 or 16. Marriage in Oriental races takes place early, and the precocity of the southern races may be due to inbreeding.

Dr. Leiner described two cases in which there had apparently been direct hereditary transmission. One patient, a girl, at birth gave the impression of a 12-year-old child; menstruation began at 6 weeks and was regular thereafter. A second patient, a boy, at 4 years of age looked as though he were at least 10,

and had the physical development of a young man of 21. The parents of both of these patients reported marked virility, or actual instances of pubertas praecox in one parent.

Precocious puberty is caused by a hypersecretion of either the gonads, pineal or cortico-suprarenal glands. A secondary involvement of the pituitary and thyroid is also unquestionable. Clinicopathologic evidence shows that the first three glands are involved in this syndrome, either in the form of hyperplasia affecting them or neoplasms.

Rogers collected 101 cases of pubertas praecox, eighty-one cases in the female, and twenty in the male; of the eighty-one cases, seventy-three point to the hyperovarian type. A number of other writers have recorded cases of this type, among them Lenz, who described the case of a girl in whom menstruation began at 16 weeks. The secondary sex characteristics were those of a mature woman. As she grew up she became a good scholar, but preferred the society of children of her own age. At 12 she was shy and childish in behavior.

Lucas reports neoplasm of the ovary as a cause of pubertas praecox. At 7 his patient showed all the signs of genitosomatic maturity, with early menses. After removal of a tumor of the ovary, all signs of adolescence and menstruation disappeared. Eleven cases of sexual precocity associated with ovarian neoplasms were collected by Roger Williams. This does not necessarily indicate that tumors of the ovary lead to sexual precocity, since other factors enter into the causation. The mentality in the ovarian cases never seems to be great, in fact, the patients speak and act their true age. Early menstruation in hyperovarianism produces excess calcium elimination. This results in short stature.

In the cortico-suprarenal type of cases the clinical picture differs according to whether the involvement is in the male or the female. Hyperplasia of the suprarenals in the male tends to accentuate male precocity; in the female the tendency is to change the female into the male type with all the secondary sexual characteristics of the male. About fourteen cases of this hypertrichosis of the male type manifested a large clitoris, complete absence of menstruation, and frequently hair on the face. The mentality in these cases is low. A certain degree of aggressiveness is reported.

In cases of hypergonadal condition in the male, mentality is usually retarded. In one case of precocious sexuality the removal of a malignant tumor of the testicle caused the disappearance of the adult characteristics.

Tumors of the pineal and their effect on sexual precocity have been extensively studied. As yet no direct connection seems to have been demonstrated, aside from the statistical fact that pineal tumors occur predominantly in the male, while those of the suprarenals are most frequent in the female.

There is little or no real mental precocity in all these types; the patients are usually shy and reserved on account of their appreciation of their differences from the normal type. Early diagnosis in the hyperplastic types may result in improvement by proper endocrine therapy.

Dr. Walter Timme added that two cases, as yet not published, had come to his attention. They were both in the same family. One of the two girls was then 16, had begun menstruating at 5 and had developed a psychosis of the dementia praecox type. The younger sister was 3 years old, menstruation had begun before 2, the child was of the incorrigible type and was apparently advanced in intelligence. Both showed pineal shadows, which are to be observed with extreme rarity at these ages. The locus of the disturbance was probably in the pineal.

With reference to Dr. Leiner's comment on the relation between skeletal growth and menstruation, Dr. Timme knew of cases that would controvert the opinion that late menstruation was accompanied by increased height. In a case that he knew, although menstruation began after 17, the patient remained small, and the epiphyses were ununited. The patient was treated on the basis of retarded growth; endocrine therapy produced rapid growth.

AN UNUSUAL CASE OF EPIDEMIC ENCEPHALOMYELITIS. Presented by DR. WALTER M. KRAUS.

J. B., a man, aged 32, a riveter, was admitted to Bellevue Hospital on Feb. 23, 1920, complaining of pains and weakness in the shoulders and arms. For three or four weeks prior to admission he had had pains in the shoulders and arms. This was increased by movements. Soon after the onset, weakness of the upper extremities became noticeable and finally compelled the patient to stop work on February 10. He noticed diplopia one week before admission to the hospital.

On admission there was weakness and tenderness of the muscles of both arms from the deltoids down. There were fibrillary twitchings in the right deltoid. There was marked winging of the right scapula (paralysis of the long respiratory nerve of Bell to the serratus magnus). Some winging of the left scapula was also present. The weakness was generally greater on the right than on the left. This may have been due to the fact that for twenty years the patient had been accustomed to carry heavy pieces of iron on the right shoulder. The pectoral muscles were strong. There was a slight weakness of the muscles supplied by the left seventh cranial nerve, and a mask-like expression. Nystagmoid movements of both eyes to right and left were observed. Tremor of the eyelids, tongue and hands was present. Reflexes: The triceps reflexes were absent; biceps ++.R<L. Supinator reflexes were present, right and left, and the other reflexes were normal. There were no sensory changes beyond the pain noted and no incoordination.

Progress: April 26: Diplopia still present. March 1: Sleepiness was marked and hard to control. March 4: Atrophy and tenderness of both intraspinati noted; white blood cells, 10,400; polymorphonuclears, 60 per cent. March 13: The tenderness in the shoulders had gradually disappeared and there was now tenderness in the hands. March 17: Pill-rolling type of tremor noticed in both hands. March 18: The gait was shuffling. Conjugate movement of both eyes downward was poor.

Laboratory Findings: Spinal fluid on admission: 40 cells; globulin +; colloidal gold curve 0000121000. The Wassermann reaction was negative.

Present Status: Slight left facial weakness; complete paralysis of the right serratus magnus and partial paralysis of the left serratus magnus (winging). Electrical Reaction: Complete reaction of degeneration in the right serratus magnus. All the other muscles of both arms, forearms and hands showed a partial reaction of degeneration. There were fibrillary tremors, atrophy and weakness of all the muscles of both upper and lower extremities. In brief: A case of acute epidemic encephalomyelitis showing among other signs the results of involvement of the anterior horn cells ofthe lower cervical (5, 6, 7, 8) and first thoracic spinal segments.

Book Review

APHASIA AND ASSOCIATED SPEECH DEFECTS. By MICHAEL OSNATO, M.D., Associate in Neurology, Columbia University. Paul B. Hoeber, New York, 1920. 183 pages.

The author of this little book is not satisfied with the doctrine of the localization of speech as that doctrine has been understood for a generation or so, and sets out to demolish it. His argument rests on two legs. First, the post-mortem findings in some cases of aphasia have not supported the claims of the localizationists. Thus, as is well known, some cases of motor aphasia have been reported in which no lesion in Broca's area had been observed. This negative proof seems to make a strong appeal to Dr. Osnato, in spite of the fact that negative proof is always open to suspicion. He makes little attempt to meet the mass of positive proof which supports the main doctrine of motor and sensory aphasia, except by reference to a few statistical papers by other workers. His own work is entirely lacking in anatomic studies. But statistical studies are usually made by the aid of the midnight oil; they are not original studies when based on papers written by others, and therefore are not of first hand value. They admit of all sorts of personal interpretation and prejudice. Anything can be proved by statistics.

The gist of the book is, of course, not in its negative or destructive criticism, but in the thesis which Dr. Osnato himself advances for the physiology of speech. He frankly returns to the antique doctrine that in the functioning of speech the brain acts as a whole. It goes off somewhat like a Leyden jar. This, we believe, or something like it, used to be the doctrine of Flourens and his school in years long gone. But Dr. Osnato marshals his pathologic data, and these he has borrowed largely from such affections as dementia praecox and other organic dementias. Using Southard's work in dementia praecox, he points out that although these patients presented speech defects, including aphasia and its associated apraxia, nevertheless the lesions in the cortex were more or less diffused and not localized in the so-called speech centers. Ergo, there is nothing in cerebral localization of speech. This argument may appeal to some emancipated critics, but it will not appeal to everybody. The simple and obvious objection to it is, of course, that a widely diffused lesion is incapable of proving anything, either pro or con, on such a subject as cerebral localization. The only proof that can stand is proof based on localized lesions —and such lesions in sufficient numbers are now on record to support the claims of the localizationists.

Dr. Osnato, summing up his opinions on page 135, repeats that "the function of speech and voluntary movement is an integral part of the development of the functions of the entire brain, and is incapable of being anatomically, physio-

logically or psychologically separated into centers." This is the whole book in a nutshell. But the writer of this review must enter a mild protest against this summary dismissal of all that we thought we had learned about cerebral localization. Let others judge for themselves of the value of Dr. Osnato's arguments by reading his book. It will entertain, even if it does not convince them.

The American Medical Association will pay 50c each for the April and May, 1919, issues of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.